SCRAPIE USA

About Me

NAME TERRY S. SINGELTARY SR. FROM ORANGE, TEXAS, UNITED STATES

My mother was murdered by what I call corporate and political homicide i.e. FOR PROFIT! she died from a rare phenotype of CJD i.e. the Heidenhain Variant of Creutzfeldt Jakob Disease i.e. sporadic, simply meaning from unknown route and source. I have simply been trying to validate her death DOD 12/14/97 with the truth. There is a route, and there is a source. There are many here in the USA. WE must make CJD and all human TSE, of all age groups 'reportable' Nationally and Internationally, with a written CJD questionnaire asking real questions pertaining to route and source of this agent. Friendly fire has the potential to play a huge role in the continued transmission of this agent via the medical, dental, and surgical arena. We must not flounder any longer. ...TSS

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MONDAY, NOVEMBER 16, 2015

Docket No. APHIS-2007-0127 Scrapie in Sheep and Goats Terry Singeltary Sr. Submission

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Docket No. APHIS-2007-0127 Scrapie in Sheep and Goats

SUMMARY: We are reopening the comment period for our proposed rule that would revise completely the scrapie regulations, which concern the risk groups and categories established for individual animals and for flocks, the use of genetic testing as a means of assigning risk levels to animals, movement restrictions for animals found to be genetically less susceptible or resistant to scrapie, and recordkeeping requirements. This action will allow interested persons additional time to prepare and submit comments.DATES: The comment period for the proposed rule published on September 10, 2015 (80 FR 54660-54692) is reopened. We will consider all comments that we receive on or before December 9, 2015. ...

http://www.regulations.gov/#!documentDetail;D=APHIS-2007-0127-0001

http://www.gpo.gov/fdsys/pkg/FR-2015-11-16/html/2015-29179.htm

http://www.regulations.gov/#!docketDetail;D=APHIS-2007-0127

COMMENT SUBMISSION TERRY S. SINGELTARY SR.

WITH regards to Docket No. APHIS-2007-0127 Scrapie in Sheep and Goats, I kindly submit the following ;

>>>The last major revision of the scrapie regulations occurred on August 21, 2001, when we published in theFederal Register(66 FR 43964, Docket No. 97-093-5) a final rule amending part 79 by imposing additional restrictions on the interstate movement of sheep and goats.<<<

Indeed, much science has changed about the Scrapie TSE prion, including more science linking Scrapie to humans. sadly, politics, industry, and trade, have not changed, and those usually trump sound science, as is the case with all Transmissible Spongiform Encephalopathy TSE Prion disease in livestock producing animals and the OIE. we can look no further at the legal trading of the Scrapie TSE prion both typical and atypical of all strains, and CWD all stains. With as much science of old, and now more new science to back this up, Scrapie of all types i.e. atypical and typical, BSE all strains, and CWD all strains, should be regulated in trade as BSE TSE PRION. In fact, I urge APHIS et al and the OIE, and all trading partners to take heed to the latest science on the TSE prion disease, all of them, and seriously reconsider the blatant disregards for human and animal health, all in the name of trade, with the continued relaxing of TSE Prion trade regulations through the 'NEGLIGIBLE BSE RISK' PROGRAM, which was set up to fail in the first place. If the world does not go back to the 'BSE RISK ASSESSMENTS', enhance, and or change that assessment process to include all TSE prion disease, i.e. 'TSE RISK ASSESSMENT', if we do not do this and if we continue this farce with OIE and the USDA et al, and the 'NEGLIGIBLE BSE RISK' PROGRAM, we will never eradicate the TSE prion aka mad cow type disease, they will

continue to mutate and spread among species of human and animal origin, and they will continue to kill. ...

please see ;

O.05: Transmission of prions to primates after extended silent incubation periods: Implications for BSE and scrapie risk assessment in human populations

Emmanuel Comoy, Jacqueline Mikol, Val erie Durand, Sophie Luccantoni, Evelyne Correia, Nathalie Lescoutra, Capucine Dehen, and Jean-Philippe Deslys Atomic Energy Commission; Fontenay-aux-Roses, France

Prion diseases (PD) are the unique neurodegenerative proteinopathies reputed to be transmissible under field conditions since decades. The transmission of Bovine Spongiform Encephalopathy (BSE) to humans evidenced that an animal PD might be zoonotic under appropriate conditions. Contrarily, in the absence of obvious (epidemiological or experimental) elements supporting a transmission or genetic predispositions, PD, like the other proteinopathies, are reputed to occur spontaneously (atpical animal prion strains, sporadic CJD summing 80% of human prion cases). Non-human primate models provided the first evidences supporting the transmissibiity of human prion strains and the zoonotic potential of BSE. Among them, cynomolgus macaques brought major information for BSE risk assessment for human health (Chen, 2014), according to their phylogenetic proximity to humans and extended lifetime. We used this model to assess the zoonotic potential of other animal PD from bovine, ovine and cervid origins even after very long silent incubation periods.

*** We recently observed the direct transmission of a natural classical scrapie isolate to macaque after a 10-year silent incubation period,

***with features similar to some reported for human cases of sporadic CJD, albeit requiring fourfold longe incubation than BSE. Scrapie, as recently evoked in humanized mice (Cassard, 2014),

***is the third potentially zoonotic PD (with BSE and L-type BSE),

***thus questioning the origin of human sporadic cases. We will present an updated panorama of our different transmission studies and discuss the implications of such extended incubation periods on risk assessment of animal PD for human health.

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thus questioning the origin of human sporadic cases

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https://prion2015.files.wordpress.com/2015/05/prion2015abstracts.pd

***Our study demonstrates susceptibility of adult cattle to oral transmission of classical BSE. ***

***our findings suggest that possible transmission risk of H-type BSE to sheep and human. ***

P.86: Estimating the risk of transmission of BSE and scrapie to ruminants and humans by protein misfolding cyclic amplification

Morikazu Imamura, Naoko Tabeta, Yoshifumi Iwamaru, and Yuichi Murayama National Institute of Animal Health; Tsukuba, Japan

To assess the risk of the transmission of ruminant prions to ruminants and humans at the molecular level, we investigated the ability of abnormal prion protein (PrPSc) of typical and atypical BSEs (L-type and H-type) and typical scrapie to convert normal prion protein (PrPC) from bovine, ovine, and human to proteinase K-resistant PrPSc-like form (PrPres) using serial protein misfolding cyclic amplification (PMCA).

Six rounds of serial PMCA was performed using 10% brain homogenates from transgenic mice expressing bovine, ovine

or human PrPC in combination with PrPSc seed from typical and atypical BSE- or typical scrapie-infected brain homogenates from native host species. In the conventional PMCA, the conversion of PrPC to PrPres was observed only when the species of PrPC source and PrPSc seed matched. However, in the PMCA with supplements (digitonin, synthetic polyA and heparin), both bovine and ovine PrPC were converted by PrPSc from all tested prion strains. On the other hand, human PrPC was converted by PrPSc from typical and H-type BSE in this PMCA condition.

Although these results were not compatible with the previous reports describing the lack of transmissibility of H-type BSE to ovine and human transgenic mice, ***our findings suggest that possible transmission risk of H-type BSE to sheep and human. Bioassay will be required to determine whether the PMCA products are infectious to these animals.

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our findings suggest that possible transmission risk of H-type BSE to sheep and human

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https://prion2015.files.wordpress.com/2015/05/prion2015abstracts.pd

https://prion2015.files.wordpress.com/2015/05/programguide1.pdf

2015

Research Project: TRANSMISSION, DIFFERENTIATION, AND PATHOBIOLOGY OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES

*** Title: Transmission of scrapie prions to primate after an extended silent incubation period Authors

item Comoy, Emmanuel - item Mikol, Jacqueline - item Luccantoni-Freire, Sophie - item Correia, Evelyne - item Lescoutra-Etchegaray, Nathalie - item Durand, Valérie - item Dehen, Capucine - item Andreoletti, Olivier - item Casalone, Cristina - item Richt, Juergen item Greenlee, Justin item Baron, Thierry - item Benestad, Sylvie - item Hills, Bob - item Brown, Paul - item Deslys, Jean-Philippe -

Submitted to: Scientific Reports Publication Type: Peer Reviewed Journal Publication Acceptance Date: May 28, 2015 Publication Date: June 30, 2015 Citation: Comoy, E.E., Mikol, J., Luccantoni-Freire, S., Correia, E., Lescoutra-Etchegaray, N., Durand, V., Dehen, C., Andreoletti, O., Casalone, C., Richt, J.A., Greenlee, J.J., Baron, T., Benestad, S., Brown, P., Deslys, J. 2015. Transmission of scrapie prions to primate after an extended silent incubation period. Scientific Reports. 5:11573. Interpretive Summary: The transmissible spongiform encephalopathies (also called prion diseases) are fatal neurodegenerative diseases that affect animals and humans. The agent of prion diseases is a misfolded form of the prion protein that is resistant to breakdown by the host cells. Since all mammals express prion protein on the surface of various cells such as neurons, all mammals are, in theory, capable of replicating prion diseases. One example of a prion disease, bovine spongiform encephalopathy (BSE; also called mad cow disease), has been shown to infect cattle, sheep, exotic undulates, cats, non-human primates, and humans when the new host is exposed to feeds or foods contaminated with the disease agent.

***The purpose of this study was to test whether non-human primates (cynomologous macaque) are susceptible to the agent of sheep scrapie. After an incubation period of approximately 10 years a macaque developed progressive clinical signs suggestive of neurologic disease. Upon postmortem examination and microscopic examination of tissues, there was a widespread distribution of lesions consistent with a transmissible spongiform encephalopathy.

***This information will have a scientific impact since it is the first study that demonstrates the transmission of scrapie to a non-human primate with a close genetic relationship to humans. This information is especially useful to regulatory officials and those involved with risk assessment of the potential transmission of animal prion diseases to humans.

Technical Abstract: Classical bovine spongiform encephalopathy (c-BSE) is an animal prion disease that also causes variant Creutzfeldt-Jakob disease in humans. Over the past decades, c-BSE's zoonotic potential has been the driving force in establishing extensive protective measures for animal and human health. In complement to the recent demonstration that humanized mice are susceptible to scrapie, we report here the first observation of direct transmission of a natural classical scrapie isolate to a macaque after a 10-year incubation period. Neuropathologic examination revealed all of the features of a prion disease: spongiform change, neuronal loss, and accumulation of PrPres throughout the CNS.

***This observation strengthens the questioning of the harmlessness of scrapie to humans, at a time when protective measures for human and animal health are being dismantled and reduced as c-BSE is considered controlled and being eradicated. Our results underscore the importance of precautionary and protective measures and the necessity for long-term experimental transmission studies to assess the zoonotic potential of other animal prion strains.

http://www.ars.usda.gov/research/publications/publications.htm?SEQ_NO_115=313160

Evidence for zoonotic potential of ovine scrapie prions

Hervé Cassard,1, n1 Juan-Maria Torres,2, n1 Caroline Lacroux,1, Jean-Yves Douet,1, Sylvie L. Benestad,3, Frédéric Lantier,4, Séverine Lugan,1, Isabelle Lantier,4, Pierrette Costes,1, Naima Aron,1, Fabienne Reine,5, Laetitia Herzog,5, Juan-Carlos Espinosa,2, Vincent Beringue5, & Olivier Andréoletti1, Affiliations Contributions Corresponding author Journal name: Nature Communications Volume: 5, Article number: 5821 DOI: doi:10.1038/ncomms6821 Received 07 August 2014 Accepted 10 November 2014 Published 16 December 2014 Article tools Citation Reprints Rights & permissions Article metrics

Abstract

Although Bovine Spongiform Encephalopathy (BSE) is the cause of variant Creutzfeldt Jakob disease (vCJD) in humans, the zoonotic potential of scrapie prions remains unknown. Mice genetically engineered to overexpress the human prion protein (tgHu) have emerged as highly relevant models for gauging the capacity of prions to transmit to humans. These models can propagate human prions without any apparent transmission barrier and have been used used to confirm the zoonotic ability of BSE. Here we show that a panel of sheep scrapie prions transmit to several tgHu mice models with an efficiency comparable to that of cattle BSE. The serial transmission of different scrapie isolates in these mice led to the propagation of prions that are phenotypically identical to those causing sporadic CJD (sCJD) in humans. These results demonstrate that scrapie prions have a zoonotic potential and raise new questions about the possible link between animal and human prions.

Subject terms: Biological sciences• Medical research At a glance

http://www.nature.com/ncomms/2014/141216/ncomms6821/full/ncomm

The serial transmission of different scrapie isolates in these mice led to the propagation of prions that are phenotypically identical to those causing sporadic CJD (sCJD) in humans.

These results demonstrate that scrapie prions have a zoonotic potential and raise new questions about the possible link between animal and human prions.

why do we not want to do TSE transmission studies on chimpanzees $

5. A positive result from a chimpanzee challenged severly would likely create alarm in some circles even if the result could not be interpreted for man. I have a view that all these agents could be transmitted provided a large enough dose by appropriate routes was given and the animals kept long enough. Until the mechanisms of the species barrier are more clearly understood it might be best to retain that hypothesis.

snip...

R. BRADLEY

http://collections.europarchive.org/tna/20080102222950/http://www.bs

1: J Infect Dis 1980 Aug;142(2):205-8

Oral transmission of kuru, Creutzfeldt-Jakob disease, and scrapie to nonhuman primates.

Gibbs CJ Jr, Amyx HL, Bacote A, Masters CL, Gajdusek DC.

Kuru and Creutzfeldt-Jakob disease of humans and scrapie disease of sheep and goats were transmitted to squirrel monkeys (Saimiri sciureus) that were exposed to the infectious agents only by their nonforced consumption of known infectious tissues. The asymptomatic incubation period in the one monkey exposed to the virus of kuru was 36 months; that in the two monkeys exposed to the virus of Creutzfeldt-Jakob disease was 23 and 27 months, respectively; and that in the two monkeys exposed to the virus of scrapie was 25 and 32 months, respectively. Careful physical examination of the buccal cavities of all of the monkeys failed to reveal signs or oral lesions. One additional monkey similarly exposed to kuru has remained asymptomatic during the 39 months that it has been under observation.

snip...

The successful transmission of kuru, Creutzfeldt-Jakob disease, and scrapie by natural feeding to squirrel monkeys that we have reported provides further grounds for concern that scrapie-infected meat may occasionally give rise in humans to Creutzfeldt-Jakob disease.

PMID: 6997404

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=6997404&dopt=Abstract

Recently the question has again been brought up as to whether scrapie is transmissible to man. This has followed reports that the disease has been transmitted to primates. One particularly lurid speculation (Gajdusek 1977) conjectures that the agents of scrapie, kuru, Creutzfeldt-Jakob disease and transmissible encephalopathy of mink are varieties of a single "virus". The U.S. Department of Agriculture concluded that it could "no longer justify or permit scrapie-blood line and scrapie-exposed sheep and goats to be processed for human or animal food at slaughter or rendering plants" (ARC 84/77)" The problem is emphasised by the finding that some strains of scrapie produce lesions identical to the once which characterise the human dementias"

Whether true or not. the hypothesis that these agents might be transmissible to man raises two considerations. First, the safety of laboratory personnel requires prompt attention. Second, action such as the "scorched meat" policy of USDA makes the solution of the acrapie problem urgent if the sheep industry is not to suffer grievously.

snip...

76/10.12/4.6

http://web.archive.org/web/20010305223125/www.bseinquiry.gov.uk/f

Nature. 1972 Mar 10;236(5341):73-4.

Transmission of scrapie to the cynomolgus monkey (Macaca fascicularis).

Gibbs CJ Jr, Gajdusek DC.

Nature 236, 73 - 74 (10 March 1972); doi:10.1038/236073a0

Transmission of Scrapie to the Cynomolgus Monkey (Macaca fascicularis)

C. J. GIBBS jun. & D. C. GAJDUSEK

National Institute of Neurological Diseases and Stroke, National Institutes of Health, Bethesda, Maryland

SCRAPIE has been transmitted to the cynomolgus, or crab-eating, monkey (Macaca fascicularis) with an incubation period of more than 5 yr from the time of intracerebral inoculation of scrapie-infected mouse brain. The animal developed a chronic central nervous system degeneration, with ataxia, tremor and myoclonus with associated severe scrapie-like pathology of intensive astroglial hypertrophy and proliferation, neuronal vacuolation and status spongiosus of grey matter. The strain of scrapie virus used was the eighth passage in Swiss mice (NIH) of a Compton strain of scrapie obtained as ninth intracerebral passage of the agent in goat brain, from Dr R. L. Chandler (ARC, Compton, Berkshire).

http://www.nature.com/nature/journal/v236/n5341/abs/236073a0.html

P03.141

Aspects of the Cerebellar Neuropathology in Nor98

Gavier-Widén, D1; Benestad, SL2; Ottander, L1; Westergren, E1 1National Veterinary Insitute, Sweden; 2National Veterinary Institute,

Norway Nor98 is a prion disease of old sheep and goats. This atypical form of scrapie was first described in Norway in 1998. Several features of Nor98 were shown to be different from classical scrapie including the distribution of disease associated prion protein (PrPd) accumulation in the brain. The cerebellum is generally the most affected brain area in Nor98. The study here presented aimed at adding information on the neuropathology in the cerebellum of Nor98 naturally affected sheep of various genotypes in Sweden and Norway. A panel of histochemical and immunohistochemical (IHC) stainings such as IHC for PrPd, synaptophysin, glial fibrillary acidic protein, amyloid, and cell markers for phagocytic cells were conducted. The type of histological lesions and tissue reactions were evaluated. The types of PrPd deposition were characterized. The cerebellar cortex was regularly affected, even though there was a variation in the severity of the lesions from case to case. Neuropil vacuolation was more marked in the molecular layer, but affected also the granular cell layer. There was a loss of granule cells. Punctate deposition of PrPd was characteristic. It was morphologically and in distribution identical with that of synaptophysin, suggesting that PrPd accumulates in the synaptic structures. PrPd was also observed in the granule cell layer and in the white matter. The pathology features of Nor98 in the cerebellum of the affected sheep showed similarities with those of sporadic Creutzfeldt-Jakob disease in humans.

***The pathology features of Nor98 in the cerebellum of the affected sheep showed similarities with those of sporadic Creutzfeldt-Jakob disease in humans.

http://www.prion2007.com/pdf/Prion%20Book%20of%20Abstracts.pd

PR-26

NOR98 SHOWS MOLECULAR FEATURES REMINISCENT OF GSS

R. Nonno1, E. Esposito1, G. Vaccari1, E. Bandino2, M. Conte1, B. Chiappini1, S. Marcon1, M. Di Bari1, S.L. Benestad3, U. Agrimi1 1 Istituto Superiore di Sanità, Department of Food Safety and Veterinary Public Health, Rome, Italy (romolo.nonno@iss.it); 2 Istituto Zooprofilattico della Sardegna, Sassari, Italy; 3 National Veterinary Institute, Department of Pathology, Oslo, Norway

Molecular variants of PrPSc are being increasingly investigated in sheep scrapie and are generally referred to as "atypical" scrapie, as opposed to "classical scrapie". Among the atypical group, Nor98 seems to be the best identified. We studied the molecular properties of Italian and Norwegian Nor98 samples by WB analysis of brain homogenates, either untreated, digested with different concentrations of proteinase K, or subjected to enzymatic deglycosylation. The identity of PrP fragments was inferred by means of antibodies spanning the full PrP sequence. We found that undigested brain homogenates contain a Nor98-specific PrP fragment migrating at 11 kDa (PrP11), truncated at both the C-terminus and the N-terminus, and not N-glycosylated. After mild PK digestion, Nor98 displayed full-length PrP (FL-PrP) and N-glycosylated C-terminal fragments (CTF), along with increased

levels of PrP11. Proteinase K digestion curves (0,006-6,4 mg/ml) showed that FL-PrP and CTF are mainly digested above 0,01 mg/ml, while PrP11 is not entirely digested even at the highest concentrations, similarly to PrP27-30 associated with classical scrapie. Above 0,2 mg/ml PK, most Nor98 samples showed only PrP11 and a fragment of 17 kDa with the same properties of PrP11, that was tentatively identified as a dimer of PrP11. Detergent solubility studies showed that PrP11 is insoluble in 2% sodium laurylsorcosine and is mainly produced from detergentsoluble, full-length PrPSc. Furthermore, among Italian scrapie isolates, we found that a sample with molecular and pathological properties consistent with Nor98 showed plaque-like deposits of PrPSc in the thalamus when the brain was analysed by PrPSc immunohistochemistry. Taken together, our results show that the distinctive pathological feature of Nor98 is a PrP fragment spanning amino acids ~ 90-155. This fragment is produced by successive N-terminal and C-terminal cleavages from a full-length and largely detergent-soluble PrPSc, is produced in vivo and is extremely resistant to PK digestion.

*** Intriguingly, these conclusions suggest that some pathological features of Nor98 are reminiscent of Gerstmann-Sträussler-Scheinker disease.

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http://www.neuroprion.com/pdf_docs/conferences/prion2006/abstract_

A newly identified type of scrapie agent can naturally infect sheep with resistant PrP genotypes

Annick Le Dur*,?, Vincent Béringue*,?, Olivier Andréoletti?, Fabienne Reine*, Thanh Lan Laï*, Thierry Baron§, Bjørn Bratberg¶, Jean-Luc Vilotte?, Pierre Sarradin**, Sylvie L. Benestad¶, and Hubert Laude*,?? +Author Affiliations

*Virologie Immunologie Moléculaires and ?Génétique Biochimique et Cytogénétique, Institut National de la Recherche Agronomique, 78350 Jouy-en-Josas, France; ? Unité Mixte de Recherche, Institut National de la Recherche Agronomique-Ecole Nationale Vétérinaire de Toulouse, Interactions Hôte Agent Pathogène, 31066 Toulouse, France; §Agence Française de Sécurité Sanitaire des Aliments, Unité Agents Transmissibles Non Conventionnels, 69364 Lyon, France; **Pathologie Infectieuse et Immunologie, Institut National de la Recherche Agronomique, 37380 Nouzilly, France; and ¶Department of Pathology, National Veterinary Institute, 0033 Oslo, Norway

***Edited by Stanley B. Prusiner, University of California, San Francisco, CA (received for review March 21, 2005)

Abstract

Scrapie in small ruminants belongs to transmissible spongiform encephalopathies (TSEs), or prion diseases, a family of fatal neurodegenerative disorders that affect humans and animals and can transmit within and between species by ingestion or inoculation. Conversion of the host-encoded prion protein (PrP), normal cellular PrP (PrPc), into a misfolded form, abnormal PrP (PrPSc), plays a key role in TSE transmission and pathogenesis. The intensified surveillance of scrapie in the European Union, together with the improvement of PrPSc detection techniques, has led to the discovery of a growing number of so-called atypical scrapie cases. These include clinical Nor98 cases first identified in Norwegian sheep on the basis of unusual pathological and PrPSc molecular features and "cases" that produced discordant responses in the rapid tests currently applied to the large-scale random screening of slaughtered or fallen animals. Worryingly, a substantial proportion of such cases involved sheep with PrP genotypes known until now to confer natural resistance to conventional scrapie. Here we report that both Nor98 and discordant cases, including three sheep homozygous for the resistant PrPARR allele (A136R154R171), efficiently transmitted the disease to transgenic mice expressing ovine PrP, and that they shared unique biological and biochemical features upon propagation in mice.

*** These observations support the view that a truly infectious TSE agent, unrecognized until recently, infects sheep and goat flocks and may have important implications in terms of scrapie

control and public health.

http://www.pnas.org/content/102/44/16031.abstract

Monday, December 1, 2008

When Atypical Scrapie cross species barriers

Authors

Andreoletti O., Herva M. H., Cassard H., Espinosa J. C., Lacroux C., Simon S., Padilla D., Benestad S. L., Lantier F., Schelcher F., Grassi J., Torres, J. M., UMR INRA ENVT 1225, Ecole Nationale Veterinaire de Toulouse.France; ICISA-INIA, Madrid, Spain; CEA, IBiTec-5, DSV, CEA/Saclay, Gif sur Yvette cedex, France; National Veterinary Institute, Postboks 750 Sentrum, 0106 Oslo, Norway, INRA IASP, Centre INRA de Tours, 3738O Nouzilly, France.

Content

Atypical scrapie is a TSE occurring in small ruminants and harbouring peculiar clinical, epidemiological and biochemical properties. Currently this form of disease is identified in a large number of countries. In this study we report the transmission of an atypical scrapie isolate through different species barriers as modeled by transgenic mice (Tg) expressing different species PRP sequence.

The donor isolate was collected in 1995 in a French commercial sheep flock. inoculation into AHQ/AHQ sheep induced a disease which had all neuro-pathological and biochemical characteristics of atypical scrapie. Transmitted into Transgenic mice expressing either ovine or PrPc, the isolate retained all the described characteristics of atypical scrapie.

Surprisingly the TSE agent characteristics were dramatically different v/hen passaged into Tg bovine mice. The recovered TSE agent had biological and biochemical characteristics similar to those of atypical BSE L in the same mouse model. Moreover, whereas no other TSE agent than BSE were shown to transmit into Tg porcine mice, atypical scrapie was able to develop into this model, albeit with low attack rate on first passage.

Furthermore, after adaptation in the porcine mouse model this prion showed similar biological and biochemical characteristics than BSE adapted to this porcine mouse model. Altogether these data indicate.

(i) the unsuspected potential abilities of atypical scrapie to cross species barriers

(ii) the possible capacity of this agent to acquire new characteristics when crossing species barrier

These findings raise some interrogation on the concept of TSE strain and on the origin of the diversity of the TSE agents and could have consequences on field TSE control measures.

http://www.neuroprion.org/resources/pdf_docs/conferences/prion2008book-prion2008.pdf

Friday, February 11, 2011

Atypical/Nor98 Scrapie Infectivity in Sheep Peripheral Tissues

http://nor-98.blogspot.com/2011/02/atypicalnor98-scrapie-infectivity-in.html

To think of Scrapie as the prime agent to compare CJD, but yet overlook the Louping-ill vaccine event in 1930's of which 1000's of sheep where infected by scrapie from a vaccine made of scrapie infected sheep brains, would be foolish. I acquired this full text version of the event which was recorded in the Annual Congress of 1946 National Vet. Med. Ass. of Great Britain and Ireland. from the BVA and the URL is posted in my (long version).

http://www.fda.gov/ohrms/dockets/ac/01/slides/3681s2_09.pdf

OR, remember the infamous Louping-ill vaccine that caused some many scrapie cases here ;

From: TSS (216-119-138-163.ipset18.wt.net)

Subject: Louping-ill vaccine documents from November 23rd, 1946

Date: September 10, 2000 at 8:57 am PST

Subject: Louping-ill vaccine documents from November 23rd, 1946

Date: Sat, 9 Sep 2000 17:44:57 –0700

From: "Terry S. Singeltary Sr."

Reply-To: Bovine Spongiform Encephalopathy

To: BSE-L@uni-karlsruhe.de

######### Bovine Spongiform Encephalopathy #########

THE VETERINARY RECORD 516 No 47. Vol. 58 November 23rd, 1946

NATIONAL VETERINARY MEDICAL ASSOCIATION OF GREAT BRITAIN AND IRELAND

ANNUAL CONGRESS, 1946

The annual Congress, 1946, was held at the Royal Veterinary College, Royal College Street, London, N.W.I. from September 22nd to September 27th.

Opening Meeting

[skip to scrapie vaccine issue...tss]

Papers Presented to Congress

The papers presented to this year's Congress had as their general theme the progressive work of the profession during the war years. Their appeal was clearly demonstrated by the large and remarkably uniform attendance in the Grand Hall of the Royal Veterinary College throughout the series; between 200 and 250 members were present and they showed a keen interest in every paper, which was reflected in the expression of some disappointment that the time available for discussion did not permit of the participation of more than a small proportion of would-be contributors.

In this issue we publish (below) the first to be read and discussed, that by Dr. W. S. Gordon, M.R.C.V.S., F.R.S.E., "Advances in Veterinary Research." Next week's issue will contain the paper on "Some Recent Advances in Veterinary Medicine and Surgery in Large-Animal Practice" by Mr. T. Norman Gold, M.R.C.V.S. In succeeding numbers of the Record will be reproduced, also with reports of discussions, that by Mr. W. L. Weipers, M.R.C.V.S., D.V.S.M., on the same subject as relating to small-animal practice, and the papers by Mr. J. N. Ritchie, B.SC., M.R.C.V.S., D.V.S.M., and Mr. H.W. Steele-Bodger, M.R.C.V.S., on "War-time Achievements of the British Home Veterinary Services."

The first scientific paper of Congress was read by Dr. W. S. Gordon, M.R.C.V.S., F.R.S.E. on Monday, September 23rd, 1946, when Professor J. Basil Buxton, M.A., F.R.C.V.S, D.V.H., Prinicipal of the Royal Veterinary College, presided.

Advances in Veterinary Research

by

W.S. GORDON, PH.D., M.R.C.V.S., F.R.S.E.

Agriculteral Research Council, Field Station, Compton, Berks.

Louping-ill, Tick-borne Fever and Scrapie

In 1930 Pool, Browniee & Wilson recorded that louping-ill was a transmissible disease. Greig et al, (1931) showed that the infective agent was a filter-passing virus with neurotropic characters and Browniee & Wilson (1932) that the essential pathology was that of an encephalomyelitis. Gordon, Browniee, Wilson & MacLeod (1932) and MacLeod & Gordon

(1932) confirmed and extended this work. It was shown that on louping-ill farms the virus was present in the blood of many sheep which did not show clinical symptoms indicating involvement of the central nervous system and that for the perpetuation and spread of the disease these subclinical cases were probably of greater importance that the frank clinical cases because, in Nature, the disease was spread by the tick, Ixodes ricinus L. More recently Wilson (1945, 1946) has described the cultivation of the virus in a chick embryo medium, the pathogenic properties of this culture virus and the preparation of louping-ill antiserum.

Between 1931 and 1934 I carried out experiments which resulted in the development of an effective vaccine for the prevention of louping-ill.* This vaccine has been in general use since 1935 and in his annual report to the Animal Diseases Research Association this year, Dr. Greig stated that about 227,000 doses of vaccine had been issued from Moredun alone.

Dr. Gordon illustrated this portion of his paper by means of graphs and diagrams projected by the epidiascope.

This investigation, however, did not begin and end with the study of louping-ill; it had, by good fortune, a more romantic turn and less fortunately a final dramtic twist which led almost to catastrope. After it had been established that a solid immunity to louping-ill could be induced in sheep, a group of immunized and a group of susceptible animals were placed together on the tick-infected pasture of a louping-ill farm. Each day all the animals were gathered and their temperatures were recorded. It was anticipated that febrile reactions with some fatalities would develop in the controls while the louping-ill immunes would remain normal. Contrary to expectation, however, every sheep, both immune and control, developed a febrile reaction. This unexpected result made neccessary further investigation which showed that the febrile reaction in the louping-ill immunes was due to a hitherto undescribed infective agent, a Rickettsia-like organism which could be observed in the cytoplasm of the grannular leucocytes, especially the neutrophil polymorphs (MacLeod (1932), Gordon, Browniee, Wilson & MacLeod. MacLeod & Gordon (1933). MacLeod (1936). MacLeod collected ticks over many widely separated parts of Scotland and all were found to harbour the infective agent of tick-borne fever, and it is probable that all sheep on tick-infested farms develop this disease, at least on the first occasion that they become infested with ticks. When the infection is passed in series through susceptible adult sheep it causes a sever, febrile reaction, dullness and loss of bodily condition but it rarely, if ever, proves fatal. It is clear, however, that it aggravates the harmful effects of a louping-ill infection and it is a serious additional complication to such infections as pyaemia and the anacrobic infections which beset lambs on the hill farms of Northern Britain.

Studying the epidemiology of louping-ill on hill farms it became obvious that the pyaemic condition of lambs described by M'Fadyean (1894) was very prevalent on tick infested farms Pyaemia is a crippling condition of lambs associated with tick-bite and is often confused with louping-ill. It is caused by infection with Staphylococcus aureus and affected animals may show abscess formation on the skin, in the joints, viscera, meninges and elsewhere in the body. It was thought that tick-borne fever might have ben a predisposing factor in this disease and unsuccessful attempts were made by Taylor, Holman & Gordon (1941) to reproduce the condition by infecting lambs subcutaneously with the staphylococcus and concurrently produceing infections with tickborne fever and louping-ill in the same lambs. Work on pyaemia was then continued by McDiarmid (1946a, 1946b, 1946c), who succeeded in reproducing a pyaemic disease in mice, guinea-pigs and lambs similar to the naturally occuring condition by intravenous inoculation of Staphylococcus aureus. He also found a bacteraemic form of the disease in which no gross pyaemic lesions were observed. The prevention or treatment of this condition presents a formidable problem. It is unlikely that staphylococcal ???oid will provide an effective immunity and even if penicillin proved to be a successful treatment, the difficulty of applying it in adequate and sustained dosage to young lambs on hill farms would be almost insurmountable.

From 1931 to 1934 field trials to test the immunizing value and harmlessness of the loup-ill vaccine were carried out on a

gradually increasing scale. Many thousands of sheep were vaccinated and similar numbers, living under identical conditions were left as controls. The end result showed that an average mortability of about 9 percent in the controls was reduced to less than 1 percent in the vaccinated animals. While the efficiency of the vaccine was obvious after the second year of work, previous bitter experience had shown the wisdom of withholding a biological product from widespread use until it had been successfully produced in bulk, as opposed to small-scale experimental production and until it had been thoroughly tested for immunizing efficiency and freedom from harmful effects. It was thought that after four years testing this stage had been reached in 1935, and in the spring of that year the vaccine was issued for general use. It comprised a 10 percent saline suspension of brain, spinal cord and spleen tissues taken from sheep five days after infection with louping-ill virus by intracerebral inoculation. To this suspension 0-35 percent of formalin was added to inactivate the virus and its safety for use as a vaccine was checked by intracerbral inoculation of mice and sheep and by the inoculation of culture medium. Its protective power was proved by vaccination sheep and later subjecting them, along with controls, to a test dose of living virus.

Vaccine for issue had to be free from detectable, living virus and capable of protecting sheep against a test dose of virus applied subcutaneously. The 1935 vaccine conformed to these standards and was issued for inoculation in March as three separate batches labelled 1, 2, and 3. The tissues of 140 sheep were employed to make batch 1 of which 22,270 doses were used; 114 to make batch 2 of which 18,000 doses were used and 44 to make batch 3 of which 4,360 doses were used. All the sheep tissues incorporated in the vaccine were obtained from yearling sheep. During 1935 and 1936 the vaccine proved highly efficient in the prevention of loup-ill and no user observed an ill-effect in the inoculated animals. In September, 1937, two and a half years after vaccinating the sheep, two owners complained that scrapie, a disease which had not before been observed in the Blackface breed, was appearing in their stock of Blackface sheep and further that it was confined to animals vaccinated with louping-ill vaccine in 1935. At that stage it was difficult to conceive that the occurrence could be associated with the injection of the vaccine but in view of the implications, I visited most of the farms on which sheep had been vaccinated in 1935. It was at this point that the investigation reached its dramatic phase; I shall not forget the profound effect on my emotions when I visited these farms and was warmly welcomed because of the great benefits resulting from the application of louping-ill vaccine, wheras the chief purpose of my visit was to determine if scrapie was appearing in the inoculated sheep. The enquiry made the position clear. Scrapie was developing in the sheep vaccinated in 1935 and it was only in a few instances that the owner was associating the occurrence with louping-ill vaccination. The disease was affecting all breeds and it was confined to the animals vaccinated with batch 2. This was clearly demonstrated on a number of farms on which batch 1 had been used to inoculate the hoggs in 1935 and batch 2 to inoculate the ewes. None of the hoggs, which at this time were three- year-old ewes. At this time it was difficult to forecast whether all of the 18,000 sheep which had received batch 2 vaccine would develop scrapie. It was fortunate, however, that the majority of the sheep vaccinated with batch 2 were ewes and therfore all that were four years old and upwards at the time of vaccination had already been disposed of and there only remained the ewes which had been two to three years old at the time of vaccination, consequently no accurate assessment of the incidence of scrapie could be made. On a few farms, however, where vaccination was confined to hoggs, the incidence ranged from 1 percent, to 35 percent, with an average of about 5 percent. Since batch 2 vaccine had been incriminated as a probable source of scrapie infection, an attempt was made to trace the origin of the 112 sheep whose tissues had been included in the vaccine. It was found that they had been supplied by three owners and that all were of the Blackface or Greyface breed with the exception of eight which were Cheviot lambs born in 1935 from ewes which had been in contact with scrapie infection. Some of these contact ewes developed scrapie in 1936-37 and three surviving fellow lambs to the eight included in the batch 2 vaccine of 1935 developed scrapie, one in September, 1936, one in February, 1937, and one in November, 1937. There was, therefore, strong presumptive evidence that the eight Cheviot lambs included in the vaccine althought apparently healthy were, in fact, in the

incubative stage of a scrapie infection and that in their tissues there was an infective agent which had contaminated the batch 2 vaccine, rendering it liable to set up scrapie. If that assumption was correct then the evidence indicated that:-

(1) the infective agent of scrapie was present in the brain, spinal cord and or spleen of infected sheep: (2) it could withstand a concentration of formalin of 0-35 percent, which inactivated the virus of louping-ill: (3) it could be transmitted by subcutaneous inoculation; (4) it had an incubative period of two years and longer.

Two Frenchmen, Cuille & Chelle (1939) as the result of experiments commenced in 1932, reported the successful infection of sheep by inoculation of emulsions of spinal cord or brain material by the intracerebral, epidural, intraocular and subcutaneous routes The incubation period varied according to the route employed, being one year intracerebrally, 15 months intraocularly and 20 months subcutaneously. They failed to infect rabbits but succeeded in infecting goats. Another important part of their work showed that the infective agent could pass throught a chamberland 1.3 filter, thus demonstrating that the infective agent was a filtrable virus. It was a curious coincidence that while they were doing their transmission experiments their work was being confirmed by the unforeseeable infectivity of a formalinized tissue vaccine.

As a result of this experience a large-scale transmision experiment involving the ue of 788 sheep was commenced in 1938 on a farm specially taken for the purpose by the Animal Diseases Research Association with funds provided by the Agricultural Research Council. The experiment was designed to determine the nature of the infective agent and the pathogenesis of the disease. It is only possible here to give a summary of the result which showed that (1) saline suspensions of brain and spinal cord tissue of sheep affected with scrapie were infective to normal sheep when inoculatted intracerebrally or subcutaneously; (2) the incubation period after intracerebral inoculation was seven months and upwards and only 60 percent of the inoculated sheep developed scrapie during a period of four and a half years; (3) the incubation period after subcutaneous inoculation was 15 months and upwards and only about 30 percent of the inoculated sheep developed the disease during the four and a half years: (4) the infective agent was of small size and probably a filtrable virus.

The prolonged incubative period of the disease and the remarkable resistance of the causal agent to formalin are features of distinct interest. It still remains to determine if a biological test can be devised to detect infected animals so that they can be killed for food before they develop clinical symptoms and to explore the possibilities of producing an immunity to the disease.
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http://nor-98.blogspot.com/2009_02_01_archive.html

the next two items are for your files, some of you might find interest...TSS

Thursday, August 20, 2015 Doctor William J. Hadlow

William J. Hadlow Dr. Hadlow (Ohio State '48), 94, Hamilton, Montana, died June 20, 2015.

http://scrapie-usa.blogspot.com/2015/08/doctor-william-j-hadlow.html

BSE: TIME TO TAKE H.B. PARRY SERIOUSLY If the scrapie agent is generated from ovine DNA and thence causes disease in other species, then perhaps, bearing in mind the possible role of scrapie in CJD of humans (Davinpour et al, 1985), scrapie and not BSE should be the notifiable disease. ...

http://collections.europarchive.org/tna/20090505194948/http://bseinqu

***2015 CWD TO HUMAN RISK FACTOR, PRICE OF POKER GOES UP ***

*** PRION 2015 CONFERENCE FT. COLLINS CWD RISK FACTORS TO HUMANS ***

*** LATE-BREAKING ABSTRACTS PRION 2015 CONFERENCE ***

O18

Zoonotic Potential of CWD Prions

Liuting Qing1, Ignazio Cali1,2, Jue Yuan1, Shenghai Huang3, Diane Kofskey1, Pierluigi Gambetti1, Wenquan Zou1, Qingzhong Kong1 1Case Western Reserve University, Cleveland, Ohio, USA, 2Second University of Naples, Naples, Italy, 3Encore Health Resources, Houston, Texas, USA

These results indicate that the CWD prion has the potential to infect human CNS and peripheral lymphoid tissues and that there might be asymptomatic human carriers of CWD infection.

P.105: RT-QuIC models trans-species prion transmission

Kristen Davenport, Davin Henderson, Candace Mathiason, and Edward Hoover Prion Research Center; Colorado State University; Fort Collins, CO USA

Additionally, human rPrP was competent for conversion by CWD and fCWD.

This insinuates that, at the level of protein:protein interactions, the barrier preventing transmission of CWD to humans is less robust than previously estimated.

https://prion2015.files.wordpress.com/2015/05/programguide1.pdf

From: Terry S. Singeltary Sr.

Sent: Saturday, November 15, 2014 9:29 PM

To: Terry S. Singeltary Sr.

Subject: THE EPIDEMIOLOGY OF CREUTZFELDT-JAKOB DISEASE R. G. WILL 1984

THE EPIDEMIOLOGY OF CREUTZFELDT-JAKOB DISEASE

R. G. WILL

1984

*** The association between venison eating and risk of CJD shows similar pattern, with regular venison eating associated with a 9 FOLD INCREASE IN RISK OF CJD (p = 0.04). (SEE LINK IN REPORT HERE...TSS) PLUS, THE CDC DID NOT PUT THIS WARNING OUT FOR THE WELL BEING OF THE DEER AND ELK ;

snip...

http://web.archive.org/web/20050425210551/http://www.bseinquiry.go

85%+ of all human tse prion disease is sporadic CJD.

see what the NIH prion Gods say themselves ;

''In the Archives of Neurology you quoted (the abstract of which was attached to your email), we did not say CWD in humans will present like variant CJD. That assumption would be wrong.''

''Also, we do not claim that "no-one has ever been infected with prion disease from eating venison." Our conclusion stating that we found no strong evidence of CWD transmission to humans in the article you quoted or in any other forum is limited to the patients we investigated.''

*** These results would seem to suggest that CWD does indeed have zoonotic potential, at least as judged by the compatibility of CWD prions and their human PrPC target. Furthermore, extrapolation from this simple in vitro assay suggests that if zoonotic CWD occurred, it would most likely effect those of the PRNP codon 129-MM genotype and that the PrPres type would be similar to that found in the most common subtype of sCJD (MM1).***

https://www.landesbioscience.com/journals/prion/article/28124/?

nocache=112223249

*** The potential impact of prion diseases on human health was greatly magnified by the recognition that interspecies transfer of BSE to humans by beef ingestion resulted in vCJD. While changes in animal feed constituents and slaughter practices appear to have curtailed vCJD, there is concern that CWD of free-ranging deer and elk in the U.S. might also cross the species barrier. Thus, consuming venison could be a source of human prion disease. Whether BSE and CWD represent interspecies scrapie transfer or are newly arisen prion diseases is unknown. Therefore, the possibility of transmission of prion disease through other food animals cannot be ruled out. There is evidence that vCJD can be transmitted through blood transfusion. There is likely a pool of unknown size of asymptomatic individuals infected with vCJD, and there may be asymptomatic individuals infected with the CWD equivalent. These circumstances represent a potential threat to blood, blood products, and plasma supplies.

http://cdmrp.army.mil/prevfunded/nprp/NPRP_Summit_Final_Report.p

now, let's see what the authors said about this casual link, personal communications years ago. see where it is stated NO STRONG evidence. so, does this mean there IS casual evidence ???? "Our conclusion stating that we found no strong evidence of CWD transmission to humans"

From: TSS (216-119-163-189.ipset45.wt.net)

Subject: CWD aka MAD DEER/ELK TO HUMANS ???

Date: September 30, 2002 at 7:06 am PST

From: "Belay, Ermias"

To: Cc: "Race, Richard (NIH)" ; ; "Belay, Ermias"

Sent: Monday, September 30, 2002 9:22 AM

Subject: RE: TO CDC AND NIH - PUB MED- 3 MORE DEATHS - CWD - YOUNG HUNTERS

Dear Sir/Madam,

In the Archives of Neurology you quoted (the abstract of which was attached to your email), we did not say CWD in humans will present like variant CJD. That assumption would be wrong. I encourage you to read the whole article and call me if you have questions or need more clarification (phone: 404-639-3091). Also, we do not claim that "no-one has ever been infected with prion disease from eating venison." Our conclusion stating that we found no strong evidence of CWD transmission to humans in the article you quoted or in any other forum is limited to the patients we investigated.

Ermias Belay, M.D. Centers for Disease Control and Prevention

-----Original Message-----

From: Sent: Sunday, September 29, 2002 10:15 AM

To: rr26k@nih.gov; rrace@niaid.nih.gov; ebb8@CDC.GOV

Subject: TO CDC AND NIH - PUB MED- 3 MORE DEATHS - CWD - YOUNG HUNTERS

Sunday, November 10, 2002 6:26 PM
.....snip.....end.....TSS

Thursday, April 03, 2008

A prion disease of cervids: Chronic wasting disease 2008 1: Vet Res. 2008 Apr 3;39(4):41 A prion disease of cervids: Chronic wasting disease Sigurdson CJ.

snip...

*** twenty-seven CJD patients who regularly consumed venison were reported to the Surveillance Center***,

snip... full text ;

http://chronic-wasting-disease.blogspot.com/2008/04/prion-disease-of-cervids-chronic.html

July's Milwaukee Journal Sentinel article did prod state officials to ask CDC to investigate the cases of the three men who shared wild game feasts. The two men the CDC is still investigating were 55 and 66 years old. But there's also Kevin Boss, a Minnesota hunter who ate Barron County venison and died of CJD at 41. And there's Jeff Schwan, whose Michigan Tech fraternity brothers used to bring venison sausage back to the frat house. His mother, Terry, says that in May 2001, Jeff, 26, began complaining about his vision. A friend noticed misspellings in his e-mail, which was totally unlike him. Jeff began losing weight. He became irritable and withdrawn. By the end of June, he couldn't remember the four-digit code to open the garage door or when and how to feed his parents' cats. At a family gathering in July, he stuck to his parents and girlfriend, barely talking. "On the night we took him to the hospital, he was speaking like he was drunk or high and I noticed his pupils were so dilated I couldn't see the irises," his mother says. By then, Jeff was no longer able to do even simple things on his computer at work, and "in the hospital, he couldn't drink enough water." When he died on September 27, 2001, an autopsy confirmed he had sporadic CJD.

In 2000, Belay looked into three CJD cases reported by The Denver Post, two hunters who ate meat from animals killed in Wyoming and the daughter of a hunter who ate venison from a plant that processed Colorado elk. All three died of CJD before they were 30 years old. The CDC asked the USDA to kill 1,000 deer and elk in the area where the men hunted. Belay and others reported their findings in the Archives of Neurology, writing that although "circumstances suggested a link between the three cases and chronic wasting disease, they could find no 'causal' link." Which means, says Belay, "not a single one of those 1,000 deer tested positive for CWD. For all we know, these cases may be CWD. What we have now doesn't indicate a connection. That's reassuring, but it would be wrong to say it will never happen."

So far, says NIH researcher Race, the two Wisconsin cases pinpointed by the newspaper look like spontaneous CJD. "But we don't know how CWD would look in human brains. It probably would look like some garden-variety sporadic CJD." What the CDC will do with these cases and four others (three from Colorado and Schwan from Upper Michigan), Race says, is "sequence the prion protein from these people, inject it into mice and wait to see what the disease looks like in their brains. That will take two years."

CJD is so rare in people under age 30, one case in a billion (leaving out medical mishaps), that four cases under 30 is "very high," says Colorado neurologist Bosque. "Then, if you add these other two from Wisconsin [cases in the newspaper], six cases of CJD in people associated with venison is very, very high." Only now, with Mary Riley, there are at least seven, and possibly eight, with Steve, her dining companion. "It's not critical mass that matters," however, Belay says. "One case would do it for me." The chance that two people who know each other would both contact CJD, like the two Wisconsin sportsmen, is so unlikely, experts say, it would happen only once in 140 years.

Given the incubation period for TSEs in humans, it may require another generation to write the final chapter on CWD in Wisconsin. "Does chronic wasting disease pass into humans? We'll be able to answer that in 2022," says Race. Meanwhile, the state has become part of an immense experiment.

https://www.organicconsumers.org/old_articles/madcow/killer123103.

I urge everyone to watch this video closely...terry

*** you can see video here and interview with Jeff's Mom, and scientist telling you to test everything and potential risk factors for humans ***

http://zoomify.uzh.ch:8080/zoomify/videos/video-004/video-004.html

*** These results would seem to suggest that CWD does indeed have zoonotic potential, at least as judged by the compatibility of CWD prions and their human PrPC target. Furthermore, extrapolation from this simple in vitro assay

suggests that if zoonotic CWD occurred, it would most likely effect those of the PRNP codon 129-MM genotype and that the PrPres type would be similar to that found in the most common subtype of sCJD (MM1).***

https://www.landesbioscience.com/journals/prion/article/28124/?nocache=112223249

Research Project: TRANSMISSION, DIFFERENTIATION, AND PATHOBIOLOGY OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES

Title: Transmission of the agent of sheep scrapie to deer results in PrPSc with two distinct molecular profiles Authors

item Greenlee, Justin item Moore, Sarah - item Smith, Jodi item West Greenlee, Mary - item Kunkle, Robert

Submitted to: Prion Publication Type: Abstract Only Publication Acceptance Date: March 31, 2015 Publication Date: May 25, 2015 Citation: Greenlee, J., Moore, S.J., Smith, J.., West Greenlee, M.H., Kunkle, R. 2015.

Scrapie transmits to white-tailed deer by the oral route and has a molecular profile similar to chronic wasting disease and distinct from the scrapie inoculum. Prion 2015. p. S62. Technical Abstract: The purpose of this work was to determine susceptibility of white-tailed deer (WTD) to the agent of sheep scrapie and to compare the resultant PrPSc to that of the original inoculum and chronic wasting disease (CWD). We inoculated WTD by a natural route of exposure (concurrent oral and intranasal (IN); n=5) with a US scrapie isolate. All scrapie-inoculated deer had evidence of PrPSc accumulation. PrPSc was detected in lymphoid tissues at preclinical time points, and deer necropsied after 28 months post-inoculation had clinical signs, spongiform encephalopathy, and widespread distribution of PrPSc in neural and lymphoid tissues. Western blotting (WB) revealed PrPSc with 2 distinct molecular profiles. WB on cerebral cortex had a profile similar to the original scrapie inoculum, whereas WB of brainstem, cerebellum, or lymph nodes reveal PrPSc with a higher profile resembling CWD. Homogenates with the 2 distinct profiles from WTD with clinical scrapie were further passaged to mice expressing cervid prion protein and intranasally to sheep and WTD. In cervidized mice, the two inocula have distinct incubation times. Sheep inoculated intranasally with WTD derived scrapie developed disease, but only after inoculation with the inoculum that had a scrapie-like profile. The WTD study is ongoing, but deer in both inoculation groups are positive for PrPSc by rectal mucosal biopsy. In summary, this work demonstrates that WTD are susceptible to the agent of scrapie, two distinct molecular profiles of PrPSc are present in the tissues of affected deer, and inoculum of either profile type readily passes to deer.

http://www.ars.usda.gov/research/publications/publications.htm?SEQ_NO_115=314097

Research Project: TRANSMISSION, DIFFERENTIATION, AND PATHOBIOLOGY OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES

Title: Scrapie transmits to white-tailed deer by the oral route and has a molecular profile similar to chronic wasting disease Authors

item Greenlee, Justin item Moore, S - item Smith, Jodi - item Kunkle, Robert item West Greenlee, M -

Submitted to: American College of Veterinary Pathologists Meeting Publication Type: Abstract Only Publication Acceptance Date: August 12, 2015 Publication Date: N/A

Technical Abstract: The purpose of this work was to determine susceptibility of white-tailed deer (WTD) to the agent of sheep scrapie and to compare the resultant PrPSc to that of the original inoculum and chronic wasting disease (CWD). We inoculated WTD by a natural route of exposure (concurrent oral and intranasal (IN); n=5) with a US scrapie isolate. All scrapie-inoculated deer had evidence of PrPSc accumulation. PrPSc was detected in lymphoid tissues at preclinical time points, and deer necropsied after 28 months post-inoculation had clinical signs, spongiform encephalopathy, and widespread distribution of PrPSc in neural and lymphoid tissues. Western

blotting (WB) revealed PrPSc with 2 distinct molecular profiles. WB on cerebral cortex had a profile similar to the original scrapie inoculum, whereas WB of brainstem, cerebellum, or lymph nodes revealed PrPSc with a higher profile resembling CWD. Homogenates with the 2 distinct profiles from WTD with clinical scrapie were further passaged to mice expressing cervid prion protein and intranasally to sheep and WTD. In cervidized mice, the two inocula have distinct incubation times. Sheep inoculated intranasally with WTD derived scrapie developed disease, but only after inoculation with the inoculum that had a scrapie-like profile. The WTD study is ongoing, but deer in both inoculation groups are positive for PrPSc by rectal mucosal biopsy. In summary, this work demonstrates that WTD are susceptible to the agent of scrapie, two distinct molecular profiles of PrPSc are present in the tissues of affected deer, and inoculum of either profile readily passes to deer.

http://www.ars.usda.gov/research/publications/publications.htm?SEQ_NO_115=317901

Sunday, October 25, 2015

USAHA Detailed Events Schedule – 119th USAHA Annual Meeting CAPTIVE LIVESTOCK CWD SCRAPIE TSE PRION

http://chronic-wasting-disease.blogspot.com/2015/10/usaha-detailed-events-schedule-119th.html

Saturday, November 14, 2015

*** TEXAS CAPTIVE BREEDER CHRONIC WASTING DISEASE CWD 2 MORE SUSPECTS DECTECTED

http://chronic-wasting-disease.blogspot.com/2015/11/texas-captive-breeder-chronic-wasting.html

Thursday, November 05, 2015

*** TPW Commission Adopts Interim Deer Breeder Movement Rules ***

http://chronic-wasting-disease.blogspot.com/2015/11/tpw-commission-adopts-interim-deer.html

Wednesday, March 18, 2015

*** Chronic Wasting Disease CWD Confirmed Texas Trans Pecos March 18, 2015 (8 cases CWD in wild to date Texas)

http://chronic-wasting-disease.blogspot.com/2015/03/chronic-wasting-disease-cwd-confirmed.html

Wednesday, March 25, 2015

*** Chronic Wasting Disease CWD Cases Confirmed In New Mexico 2013 and 2014 UPDATE 2015

http://chronic-wasting-disease.blogspot.com/2015/03/chronic-wasting-disease-cwd-cases.html

*** Spraker suggested an interesting explanation for the occurrence of CWD. The deer pens at the Foot Hills Campus were built some 30-40 years ago by a Dr. Bob Davis. At or abut that time, allegedly, some scrapie work was conducted at this site. When deer were introduced to the pens they occupied ground that had previously been occupied by sheep.

http://collections.europarchive.org/tna/20080102193705/http://www.bs

White-tailed Deer are Susceptible to Scrapie by Natural Route of Infection

Jodi D. Smith, Justin J. Greenlee, and Robert A. Kunkle; Virus and Prion Research Unit, National Animal Disease Center, USDA-ARS

Interspecies transmission studies afford the opportunity to better understand the potential host range and origins of prion diseases. Previous experiments demonstrated that white-tailed deer are susceptible to sheep-derived scrapie by intracranial inoculation. The purpose of this study was to determine susceptibility of white-tailed deer to scrapie after a natural route of exposure. Deer (n=5) were inoculated by

concurrent oral (30 ml) and intranasal (1 ml) instillation of a 10% (wt/vol) brain homogenate derived from a sheep clinically affected with scrapie. Non-inoculated deer were maintained as negative controls. All deer were observed daily for clinical signs. Deer were euthanized and necropsied when neurologic disease was evident, and tissues were examined for abnormal prion protein (PrPSc) by immunohistochemistry (IHC) and western blot (WB). One animal was euthanized 15 months post-inoculation (MPI) due to an injury. At that time, examination of obex and lymphoid tissues by IHC was positive, but WB of obex and colliculus were negative. Remaining deer developed clinical signs of wasting and mental depression and were necropsied from 28 to 33 MPI. Tissues from these deer were positive for scrapie by IHC and WB. Tissues with PrPSc immunoreactivity included brain, tonsil, retropharyngeal and mesenteric lymph nodes, hemal node, Peyer's patches, and spleen. This work demonstrates for the first time that white-tailed deer are susceptible to sheep scrapie by potential natural routes of inoculation. In-depth analysis of tissues will be done to determine similarities between scrapie in deer after intracranial and oral/intranasal inoculation and chronic wasting disease resulting from similar routes of inoculation.

see full text ;

http://www.usaha.org/Portals/6/Reports/2010/report-cwal-2010.pdf

PO-039: A comparison of scrapie and chronic wasting disease in white-tailed deer

Justin Greenlee, Jodi Smith, Eric Nicholson US Dept. Agriculture; Agricultural Research Service, National Animal Disease Center; Ames, IA USA

http://www.landesbioscience.com/journals/prion/03-Prion6-2-Transmission-and-strains.pdf

White-tailed deer are susceptible to the agent of sheep scrapie by intracerebral inoculation

snip...

It is unlikely that CWD will be eradicated from free-ranging cervids, and the disease is likely to continue to spread geographically [10]. However, the potential that white-tailed deer may be susceptible to sheep scrapie by a natural route presents an additional confounding factor to halting the spread of CWD. This leads to the additional speculations that

1) infected deer could serve as a reservoir to infect sheep with scrapie offering challenges to scrapie eradication efforts and

2) CWD spread need not remain geographically confined to current endemic areas, but could occur anywhere that sheep with scrapie and susceptible cervids cohabitate.

This work demonstrates for the first time that white-tailed deer are susceptible to sheep scrapie by intracerebral inoculation with a high attack rate and that the disease that results has similarities to CWD. These experiments will be repeated with a more natural route of inoculation to determine the likelihood of the potential transmission of sheep scrapie to white-tailed deer. If scrapie were to occur in white-tailed deer, results of this study indicate that it would be detected as a TSE, but may be difficult to differentiate from CWD without in-depth biochemical analysis.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3199251/?tool=pubmed

http://chronic-wasting-disease.blogspot.com/2011/10/white-tailed-deer-are-susceptible-to.html

2012

PO-039: A comparison of scrapie and chronic wasting disease in white-tailed deer

Justin Greenlee, Jodi Smith, Eric Nicholson US Dept. Agriculture; Agricultural Research Service, National Animal Disease Center; Ames, IA USA

snip...

The results of this study suggest that there are many similarities in the manifestation of CWD and scrapie in WTD after IC inoculation including early and widespread presence of PrPSc in lymphoid tissues, clinical signs of depression and weight loss progressing to wasting, and an incubation time of 21-23 months. Moreover, western blots (WB) done on brain material from the obex region have a molecular profile similar to CWD and distinct from tissues of the cerebrum or the scrapie inoculum. However, results of microscopic and IHC examination indicate that there are differences between the lesions expected in CWD and those that occur in deer with scrapie: amyloid plaques were not noted in any sections of brain examined from these deer and the pattern of immunoreactivity by IHC was diffuse rather than plaque-like.

*** After a natural route of exposure, 100% of WTD were susceptible to scrapie.

Deer developed clinical signs of wasting and mental depression and were necropsied from 28 to 33 months PI. Tissues from these deer were positive for PrPSc by IHC and WB. Similar to IC inoculated deer, samples from these deer exhibited two different molecular profiles: samples from obex resembled CWD whereas those from cerebrum were similar to the original scrapie inoculum. On further examination by WB using a panel of antibodies, the tissues from deer with scrapie exhibit properties differing from tissues either from sheep with scrapie or WTD with CWD. Samples from WTD with CWD or sheep with scrapie are strongly immunoreactive when probed with mAb P4, however, samples from WTD with scrapie are only weakly immunoreactive. In contrast, when probed with mAb's 6H4 or SAF 84, samples from sheep with scrapie and WTD with CWD are weakly immunoreactive and samples from WTD with scrapie are strongly positive. This work demonstrates that WTD are highly susceptible to sheep scrapie, but on first passage, scrapie in WTD is differentiable from CWD.

http://www.landesbioscience.com/journals/prion/03-Prion6-2-Transmission-and-strains.pdf

2011

*** After a natural route of exposure, 100% of white-tailed deer were susceptible to scrapie.

http://www.usaha.org/Portals/6/Reports/2011/report-cwal-2011.pdf

Sunday, October 25, 2015

USAHA Detailed Events Schedule – 119th USAHA Annual Meeting CAPTIVE LIVESTOCK CWD SCRAPIE TSE PRION

http://chronic-wasting-disease.blogspot.com/2015/10/usaha-detailed-events-schedule-119th.html

PL1

Using in vitro prion replication for high sensitive detection of prions and prionlike proteins and for understanding mechanisms of transmission.

Claudio Soto

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Prion and prion-like proteins are misfolded protein aggregates with the ability to selfpropagate to spread disease between cells, organs and in some cases across individuals. In Transmissible spongiform encephalopathies (TSEs), prions are mostly composed by a misfolded form of the prion protein (PrPSc), which propagates by transmitting its misfolding to the normal prion protein (PrPC). The availability of a procedure to replicate prions in the laboratory may be important to study the mechanism of prion and prion-like spreading and to develop high sensitive detection of small quantities of misfolded proteins in biological fluids, tissues and environmental samples. Protein Misfolding Cyclic Amplification (PMCA) is a simple, fast and efficient

methodology to mimic prion replication in the test tube. PMCA is a platform technology that may enable amplification of any prion-like misfolded protein aggregating through a seeding/nucleation process. In TSEs, PMCA is able to detect the equivalent of one single molecule of infectious PrPSc and propagate prions that maintain high infectivity, strain properties and species specificity. Using PMCA we have been able to detect PrPSc in blood and urine of experimentally infected animals and humans affected by vCJD with high sensitivity and specificity. Recently, we have expanded the principles of PMCA to amplify amyloid-beta (Aβ) and alphasynuclein (α-syn) aggregates implicated in Alzheimer's and Parkinson's diseases, respectively. Experiments are ongoing to study the utility of this technology to detect Aβ and α-syn aggregates in samples of CSF and blood from patients affected by these diseases.

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***Recently, we have been using PMCA to study the role of environmental prion contamination on the horizontal spreading of TSEs. These experiments have focused on the study of the interaction of prions with plants and environmentally relevant surfaces. Our results show that plants (both leaves and roots) bind tightly to prions present in brain extracts and excreta (urine and feces) and retain even small quantities of PrPSc for long periods of time. Strikingly, ingestion of prioncontaminated leaves and roots produced disease with a 100% attack rate and an incubation period not substantially longer than feeding animals directly with scrapie brain homogenate. Furthermore, plants can uptake prions from contaminated soil and transport them to different parts of the plant tissue (stem and leaves). Similarly, prions bind tightly to a variety of environmentally relevant surfaces, including stones, wood, metals, plastic, glass, cement, etc. Prion contaminated surfaces efficiently transmit prion disease when these materials were directly injected into the brain of animals and strikingly when the contaminated surfaces were just placed in the animal cage. These findings demonstrate that environmental materials can efficiently bind infectious prions and act as carriers of infectivity, suggesting that they may play an important role in the horizontal transmission of the disease.

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Since its invention 13 years ago, PMCA has helped to answer fundamental questions of prion propagation and has broad applications in research areas including the food industry, blood bank safety and human and veterinary disease diagnosis.

https://prion2015.files.wordpress.com/2015/05/programguide1.pdf

see ;

http://www.tandfonline.com/doi/pdf/10.4161/pri.28467

http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0058630

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3567181/pdf/ppat.10031

http://www.nature.com/srep/2015/150210/srep08358/full/srep08358.ht
WT.ec_id=SREP-639-20150217

http://www.cell.com/cell-reports/pdfExtended/S2211-1247(15)00437-4

98 | Veterinary Record | January 24, 2015

EDITORIAL

Scrapie: a particularly persistent pathogen

Cristina Acín

Resistant prions in the environment have been the sword of Damocles for scrapie control and eradication. Attempts to establish which physical and chemical agents could be applied to inactivate or moderate scrapie infectivity were initiated in the 1960s and 1970s,with the first study of this type focusing on the effect of heat treatment in reducing prion infectivity (Hunter and Millson 1964). Nowadays, most of the chemical procedures that aim to inactivate the prion protein

are based on the method developed by Kimberlin and collaborators (1983). This procedure consists of treatment with 20,000 parts per million free chlorine solution, for a minimum of one hour, of all surfaces that need to be sterilised (in laboratories, lambing pens, slaughterhouses, and so on). Despite this, veterinarians and farmers may still ask a range of questions, such as 'Is there an official procedure published somewhere?' and 'Is there an international organisation which recommends and defines the exact method of scrapie decontamination that must be applied?'

From a European perspective, it is difficult to find a treatment that could be applied, especially in relation to the disinfection of surfaces in lambing pens of affected flocks. A 999/2001 EU regulation on controlling spongiform encephalopathies (European Parliament and Council 2001) did not specify a particular decontamination measure to be used when an outbreak of scrapie is diagnosed. There is only a brief recommendation in Annex VII concerning the control and eradication of transmissible spongiform encephalopathies (TSE s).

Chapter B of the regulation explains the measures that must be applied if new caprine animals are to be introduced to a holding where a scrapie outbreak has previously been diagnosed. In that case, the statement indicates that caprine animals can be introduced 'provided that a cleaning and disinfection of all animal housing on the premises has been carried out following destocking'.

Issues around cleaning and disinfection are common in prion prevention recommendations, but relevant authorities, veterinarians and farmers may have difficulties in finding the specific protocol which applies. The European Food and Safety Authority (EFSA) published a detailed report about the efficacy of certain biocides, such as sodium hydroxide, sodium hypochlorite, guanidine and even a formulation of copper or iron metal ions in combination with hydrogen peroxide, against prions (EFSA 2009). The report was based on scientific evidence (Fichet and others 2004, Lemmer and others 2004, Gao and others 2006, Solassol and others 2006) but unfortunately the decontamination measures were not assessed under outbreak conditions.

The EFSA Panel on Biological Hazards recently published its conclusions on the scrapie situation in the EU after 10 years of monitoring and control of the disease in sheep and goats (EFSA 2014), and one of the most interesting findings was the Icelandic experience regarding the effect of disinfection in scrapie control. The Icelandic plan consisted of: culling scrapie-affected sheep or the whole flock in newly diagnosed outbreaks; deep cleaning and disinfection of stables, sheds, barns and equipment with high pressure washing followed by cleaning with 500 parts per million of hypochlorite; drying and treatment with 300 ppm of iodophor; and restocking was not permitted for at least two years. Even when all of these measures were implemented, scrapie recurred on several farms, indicating that the infectious agent survived for years in the environment, even as many as 16 years after restocking (Georgsson and others 2006).

In the rest of the countries considered in the EFSA (2014) report, recommendations for disinfection measures were not specifically defined at the government level. In the report, the only recommendation that is made for sheep is repopulation with sheep with scrapie-resistant genotypes. This reduces the risk of scrapie recurrence but it is difficult to know its effect on the infection.

Until the EFSA was established (in May 2003), scientific opinions about TSE s were provided by the Scientific Steering Committee (SSC) of the EC, whose advice regarding inactivation procedures focused on treating animal waste at high temperatures (150°C for three hours) and high pressure alkaline hydrolysis (SSC 2003). At the same time, the TSE Risk Management Subgroup of the Advisory Committee on Dangerous Pathogens (ACDP) in the UK published guidance on safe working and the prevention of TSE infection. Annex C of the ACDP report established that sodium hypochlorite was considered to be effective, but only if 20,000 ppm of available chlorine was present for at least one hour, which has practical limitations such as the release of chlorine gas, corrosion, incompatibility with formaldehyde, alcohols and acids, rapid inactivation of its active chemicals and the stability of dilutions

(ACDP 2009).

In an international context, the World Organisation for Animal Health (OIE) does not recommend a specific disinfection protocol for prion agents in its Terrestrial Code or Manual. Chapter 4.13 of the Terrestrial Code, General recommendations on disinfection and disinsection (OIE 2014), focuses on foot-and-mouth disease virus, mycobacteria and Bacillus anthracis, but not on prion disinfection. Nevertheless, the last update published by the OIE on bovine spongiform encephalopathy (OIE 2012) indicates that few effective decontamination techniques are available to inactivate the agent on surfaces, and recommends the removal of all organic material and the use of sodium hydroxide, or a sodium hypochlorite solution containing 2 per cent available chlorine, for more than one hour at 20ºC.

The World Health Organization outlines guidelines for the control of TSE s, and also emphasises the importance of mechanically cleaning surfaces before disinfection with sodium hydroxide or sodium hypochlorite for one hour (WHO 1999).

Finally, the relevant agencies in both Canada and the USA suggest that the best treatments for surfaces potentially contaminated with prions are sodium hydroxide or sodium hypochlorite at 20,000 ppm. This is a 2 per cent solution, while most commercial household bleaches contain 5.25 per cent sodium hypochlorite. It is therefore recommended to dilute one part 5.25 per cent bleach with 1.5 parts water (CDC 2009, Canadian Food Inspection Agency 2013).

So what should we do about disinfection against prions? First, it is suggested that a single protocol be created by international authorities to homogenise inactivation procedures and enable their application in all scrapie-affected countries. Sodium hypochlorite with 20,000 ppm of available chlorine seems to be the procedure used in most countries, as noted in a paper summarised on p 99 of this issue of Veterinary Record (Hawkins and others 2015). But are we totally sure of its effectiveness as a preventive measure in a scrapie outbreak? Would an in-depth study of the recurrence of scrapie disease be needed?

What we can conclude is that, if we want to fight prion diseases, and specifically classical scrapie, we must focus on the accuracy of diagnosis, monitoring and surveillance; appropriate animal identification and control of movements; and, in the end, have homogeneous and suitable protocols to decontaminate and disinfect lambing barns, sheds and equipment available to veterinarians and farmers. Finally, further investigations into the resistance of prion proteins in the diversity of environmental surfaces are required.

References

snip...

98 | Veterinary Record | January 24, 2015

http://veterinaryrecord.bmj.com/content/176/4/97.extract

*** Infectious agent of sheep scrapie may persist in the environment for at least 16 years ***

Gudmundur Georgsson1, Sigurdur Sigurdarson2 and Paul Brown3

http://jgv.sgmjournals.org/content/87/12/3737.full

Persistence of ovine scrapie infectivity in a farm environment following cleaning and decontamination

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Leicestershire LE12 5RD, UK E-mail for correspondence: ben.maddison@adas.co.uk Abstract Scrapie of sheep/goats and chronic wasting disease of deer/elk are contagious prion diseases where environmental reservoirs are directly implicated in the transmission of disease. In this study, the effectiveness of recommended scrapie farm decontamination regimens was evaluated by a sheep bioassay using buildings naturally contaminated with scrapie. Pens within a farm building were treated with either 20,000 parts per million free chorine solution for one hour or were treated with the same but were followed by painting and full re-galvanisation or replacement of metalwork within the pen. Scrapie susceptible lambs of the PRNP genotype VRQ/VRQ were reared within these pens and their scrapie status was monitored by recto-anal mucosa-associated lymphoid tissue. All animals became infected over an 18-month period, even in the pen that had been subject to the most stringent decontamination process. These data suggest that recommended current guidelines for the decontamination of farm buildings following outbreaks of scrapie do little to reduce the titre of infectious scrapie material and that environmental recontamination could also be an issue associated with these premises.

SNIP...

Discussion

Thorough pressure washing of a pen had no effect on the amount of bioavailable scrapie infectivity (pen B). The routine removal of prions from surfaces within a laboratory setting is treatment for a minimum of one hour with 20,000 ppm free chlorine, a method originally based on the use of brain macerates from infected rodents to evaluate the effectiveness of decontamination (Kimberlin and others 1983). Further studies have also investigated the effectiveness of hypochlorite disinfection of metal surfaces to simulate the decontamination of surgical devices within a hospital setting. Such treatments with hypochlorite solution were able to reduce infectivity by 5.5 logs to lower than the sensitivity of the bioassay used (Lemmer and others 2004). Analogous treatment of the pen surfaces did not effectively remove the levels of scrapie infectivity over that of the control pens, indicating that this method of decontamination is not effective within a farm setting. This may be due to the high level of biological matrix that is present upon surfaces within the farm environment, which may reduce the amount of free chlorine available to inactivate any infectious prion. Remarkably 1/5 sheep introduced into pen D had also became scrapie positive within nine months, with all animals in this pen being RAMALT positive by 18 months of age. Pen D was no further away from the control pen (pen A) than any of the other pens within this barn. Localised hot spots of infectivity may be present within scrapie-contaminated environments, but it is unlikely that pen D area had an amount of scrapie contamination that was significantly different than the other areas within this building. Similarly, there were no differences in how the biosecurity of pen D was maintained, or how this pen was ventilated compared with the other pens. This observation, perhaps, indicates the slower kinetics of disease uptake within this pen and is consistent with a more thorough prion removal and recontamination. These observations may also account for the presence of inadvertent scrapie cases within other studies, where despite stringent biosecurity, control animals have become scrapie positive during challenge studies using barns that also housed scrapie-affected animals (Ryder and others 2009).

***The bioassay data indicate that the exposure of the sheep to a farm environment after decontamination efforts thought to be effective in removing scrapie is sufficient for the animals to become infected with scrapie. The main exposure routes within this scenario are likely to be via the oral route, during feeding and drinking, and respiratory and conjunctival routes. It has been demonstrated that scrapie infectivity can be efficiently transmitted via the nasal route in sheep (Hamir and others 2008), as is the case for CWD in both murine models and in white-tailed deer (Denkers and others 2010, 2013).

Recently, it has also been demonstrated that CWD prions presented as dust when bound to the soil mineral montmorillonite can be infectious via the nasal route (Nichols and others 2013). When considering pens C and D, the actual source of the infectious agent in the pens is not known, it is possible that biologically relevant levels of prion survive on

surfaces during the decontamination regimen (pen C). With the use of galvanising and painting (pen D) covering and sealing the surface of the pen, it is possible that scrapie material recontaminated the pens by the movement of infectious prions contained within dusts originating from other parts of the barn that were not decontaminated or from other areas of the farm.

Given that scrapie prions are widespread on the surfaces of affected farms (Maddison and others 2010a), irrespective of the source of the infectious prions in the pens, this study clearly highlights the difficulties that are faced with the effective removal of environmentally associated scrapie infectivity. This is likely to be paralleled in CWD which shows strong similarities to scrapie in terms of both the dissemination of prions into the environment and the facile mode of disease transmission. These data further contribute to the understanding that prion diseases can be highly transmissible between susceptible individuals not just by direct contact but through highly stable environmental reservoirs that are refractory to decontamination.

The presence of these environmentally associated prions in farm buildings make the control of these diseases a considerable challenge, especially in animal species such as goats where there is lack of genetic resistance to scrapie and, therefore, no scope to re-stock farms with animals that are resistant to scrapie.

Scrapie Sheep Goats Transmissible spongiform encephalopathies (TSE) Accepted October 12, 2014. Published Online First 31 October 2014

http://veterinaryrecord.bmj.com/content/early/2014/10/31/vr.102743.ab

Monday, November 3, 2014

Persistence of ovine scrapie infectivity in a farm environment following cleaning and decontamination

http://transmissiblespongiformencephalopathy.blogspot.com/2014/11/
of-ovine-scrapie.html

PPo3-22:

Detection of Environmentally Associated PrPSc on a Farm with Endemic Scrapie

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Key words: scrapie, evironmental persistence, sPMCA

Ovine scrapie shows considerable horizontal transmission, yet the routes of transmission and specifically the role of fomites in transmission remain poorly defined. Here we present biochemical data demonstrating that on a scrapie-affected sheep farm, scrapie prion contamination is widespread. It was anticipated at the outset that if prions contaminate the environment that they would be there at extremely low levels, as such the most sensitive method available for the detection of PrPSc, serial Protein Misfolding Cyclic Amplification (sPMCA), was used in this study. We investigated the distribution of environmental scrapie prions by applying ovine sPMCA to samples taken from a range of surfaces that were accessible to animals and could be collected by use of a wetted foam swab. Prion was amplified by sPMCA from a number of these environmental swab samples including those taken from metal, plastic and wooden surfaces, both in the indoor and outdoor environment. At the time of sampling there had been no sheep contact with these areas for at least 20 days prior to sampling indicating that prions persist for at least this duration in the environment. These data implicate inanimate objects as environmental reservoirs of prion infectivity which are likely to contribute to disease transmission.

http://www.prion2010.org/bilder/prion_2010_program_latest_w_poste
139&PHPSESSID=a30a38202cfec579000b77af81be3099

>>>We report here the identification and characterization of 2 natural classic scrapie cases in sheep of the ARR/ARR genotype, which are clearly different from BSE and atypical scrapie.

Classic Scrapie in Sheep with the ARR/ARR Prion Genotype in Germany and France

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In the past, natural scrapie and bovine spongiform encephalopathy (BSE) infections have essentially not been diagnosed in sheep homozygous for the A136R154R171 haplotype of the prion protein. This genotype was therefore assumed to confer resistance to BSE and classic scrapie under natural exposure conditions. Hence, to exclude prions from the human food chain, massive breeding efforts have been undertaken in the European Union to amplify this gene. We report the identifi cation of 2 natural scrapie cases in ARR/ARR sheep that have biochemical and transmission characteristics similar to cases of classic scrapie, although the abnormally folded prion protein (PrPSc) was associated with a lower proteinase-K resistance. PrPSc was clearly distinct from BSE prions passaged in sheep and from atypical scrapie prions. These findings strongly support the idea that scrapie prions are a mosaic of agents, which harbor different biologic properties, rather than a unique entity.

snip...

However, the successful transmission of BSE prions to ARR/ARR sheep showed that the resistance of this genotype toward the TSE agent was not absolute (11). Recently, the identification of previously unrecognized so-called atypical scrapie in sheep of various genotypes, including ARR/ARR, has reinforced this statement (4).

***We report here the identification and characterization of 2 natural classic scrapie cases in sheep of the ARR/ARR genotype, which are clearly different from BSE and atypical scrapie.

snip...

see full text ;

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2828083/pdf/07-0077_finalR.pdf

Assessing Transmissible Spongiform Encephalopathy Species Barriers with an In Vitro Prion Protein Conversion Assay

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Summary

Measuring the barrier to the interspecies transmission of prion diseases is challenging and typically involves animal challenges or biochemical assays. Here, we present an in vitro prion protein conversion assay with the ability to predict species barriers.

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Keywords: Medicine, Issue 97, Prion, species barrier, conversion, immunoblotting, transmissible spongiform encephalopathy, interspecies transmission Cite this Article

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Cashman, N. R. Assessing Transmissible Spongiform Encephalopathy Species Barriers with an In Vitro Prion Protein Conversion Assay. J. Vis. Exp. (97), e52522, doi:10.3791/52522 (2015). Abstract

Studies to understanding interspecies transmission of transmissible spongiform encephalopathies (TSEs, prion diseases) are challenging in that they typically rely upon lengthy and costly in vivo animal challenge studies. A number of in vitro assays have been developed to aid in measuring prion species barriers, thereby reducing animal use and providing quicker results than animal bioassays. Here, we present the protocol for a rapid in vitro prion conversion assay called the conversion efficiency ratio (CER) assay. In this assay cellular prion protein (PrPC) from an uninfected host brain is denatured at both pH 7.4 and 3.5 to produce two substrates. When the pH 7.4 substrate is incubated with TSE agent, the amount of PrPC that converts to a proteinase K (PK)-resistant state is modulated by the original host's species barrier to the TSE agent. In contrast, PrPC in the pH 3.5 substrate is misfolded by any TSE agent. By comparing the amount of PK-resistant prion protein in the two substrates, an assessment of the host's species barrier can be made. We show that the CER assay correctly predicts known prion species barriers of laboratory mice and, as an example, show some preliminary results suggesting that bobcats (Lynx rufus) may be susceptible to white-tailed deer (Odocoileus virginianus) chronic wasting disease agent.

http://www.jove.com/video/52522/assessing-transmissible-spongiform-encephalopathy-species-barriers

>>> show some preliminary results suggesting that bobcats (Lynx rufus) may be susceptible to white-tailed deer (Odocoileus virginianus) chronic wasting disease agent.

AD.63: Susceptibility of domestic cats to chronic wasting disease

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Domestic and nondomestic cats have been shown to be susceptible to feline spongiform encephalopathy (FSE), almost certainly caused by consumption of bovine spongiform encephalopathy (BSE)-contaminated meat. Because domestic and free-ranging nondomestic felids scavenge cervid carcasses, including those in areas affected by chronic wasting disease (CWD), we evaluated the susceptibility of the domestic cat (Felis catus) to CWD infection experimentally. Cohorts of 5 cats each were inoculated either intracerebrally (IC) or orally (PO) with CWD-infected deer brain. At 40 and 42 mo post-inoculation, two IC-inoculated cats developed signs consistent with prion disease, including a stilted gait, weight loss, anorexia, polydipsia, patterned motor behaviors, head and tail tremors, and ataxia, and progressed to terminal disease within 5 mo. Brains from these two cats were pooled and inoculated into cohorts of cats by IC, PO, and intraperitoneal and subcutaneous (IP/SC) routes. Upon subpassage, feline-adapted CWD (FelCWD) was transmitted to all IC-inoculated cats with a decreased incubation period of 23 to 27 mo. FelCWD was detected in the brains of all the symptomatic cats by western blotting and immunohistochemistry and abnormalities were seen in magnetic resonance imaging, including multifocal T2 fluid attenuated inversion recovery (FLAIR) signal hyper-intensities, ventricular size increases, prominent sulci, and white matter tract cavitation. Currently, 3 of 4 IP/SQ and 2 of 4 PO inoculared cats have developed abnormal behavior patterns consistent with the early stage of feline CWD. These results demonstrate that CWD can be transmitted and adapted to the domestic cat, thus raising the issue of potential cervid-to-feline transmission in nature.

http://www.prion2013.ca/tiny_uploads/forms/Scientific-Program.pdf

www.landesbioscience.com

PO-081: Chronic wasting disease in the cat— Similarities to

feline spongiform encephalopathy (FSE)

http://www.landesbioscience.com/journals/prion/04-Prion6-2-Pathogenesis-and-pathology.pdf

http://chronic-wasting-disease.blogspot.com/2012/05/chronic-wasting-disease-cwd-prion2012.html

http://www.prion2011.ca/files/PRION_2011_-_Posters_(May_5-11).pdf

http://felinespongiformencephalopathyfse.blogspot.com/2011/08/susce of-domestic-cats-to-cwd.html

PO-081: Chronic wasting disease in the cat— Similarities to feline spongiform encephalopathy (FSE)

http://www.landesbioscience.com/journals/prion/04-Prion6-2-Pathogenesis-and-pathology.pdf

http://chronic-wasting-disease.blogspot.com/2012/05/chronic-wasting-disease-cwd-prion2012.html

Thursday, May 31, 2012

CHRONIC WASTING DISEASE CWD PRION2012 Aerosol, Inhalation transmission, Scrapie, cats, species barrier, burial, and more

http://chronic-wasting-disease.blogspot.com/2012/05/chronic-wasting-disease-cwd-prion2012.html

Monday, August 8, 2011

Susceptibility of Domestic Cats to CWD Infection

http://felinespongiformencephalopathyfse.blogspot.com/2011/08/susce of-domestic-cats-to-cwd.html

Sunday, August 25, 2013

Prion2013 Chronic Wasting Disease CWD risk factors, humans, domestic cats, blood, and mother to offspring transmission

http://chronic-wasting-disease.blogspot.com/2013/08/prion2013-chronic-wasting-disease-cwd.html

Feline Spongiform Encephalopathy (FSE) FSE was first identified in the UK in 1990. Most cases have been reported in the UK, where the epidemic has been consistent with that of the BSE epidemic. Some other countries (e.g. Norway, Liechtenstein and France) have also reported cases.

Most cases have been reported in domestic cats but there have also been cases in captive exotic cats (e.g. Cheetah, Lion, Asian leopard cat, Ocelot, Puma and Tiger). The disease is characterised by progressive nervous signs, including ataxia, hyper-reactivity and behavioural changes and is fatal.

The chemical and biological properties of the infectious agent are identical to those of the BSE and vCJD agents. These findings support the hypothesis that the FSE epidemic resulted from the consumption of food contaminated with the BSE agent.

The FSE epidemic has declined as a result of tight controls on the disposal of specified risk material and other animal by-products.

References: Leggett, M.M. et al.(1990) A spongiform encephalopathy in a cat. Veterinary Record. 127. 586-588

Synge, B.A. et al. (1991) Spongiform encephalopathy in a Scottish cat. Veterinary Record. 129. 320

Wyatt, J. M. et al. (1991) Naturally occurring scrapie-like spongiform encephalopathy in five domestic cats. Veterinary Record. 129. 233.

Gruffydd-Jones, T. J.et al.. (1991) Feline spongiform encephalopathy. J. Small Animal Practice. 33. 471-476.

Pearson, G. R. et al. (1992) Feline spongiform encephalopathy: fibril and PrP studies. Veterinary Record. 131. 307-310.

Willoughby, K. et al. (1992) Spongiform encephalopathy in a captive puma (Felis concolor). Veterinary Record. 131. 431-434.

Fraser, H. et al. (1994) Transmission of feline spongiform encephalopathy to mice. Veterinary Record 134. 449.

Bratberg, B. et al. (1995) Feline spongiform encephalopathy in a cat in Norway. Veterinary Record 136. 444

Baron, T. et al. (1997) Spongiform encephalopathy in an imported cheetah in France. Veterinary Record 141. 270-271

Zanusso, G et al. (1998) Simultaneous occurrence of spongiform encephalopathy in a man and his cat in Italy. Lancet, V352, N9134, OCT 3, Pp 1116-1117.

Ryder, S.J. et al. (2001) Inconsistent detection of PrP in extraneural tissues of cats with feline spongiform encephalopathy. Veterinary Record 146. 437-441

Kelly, D.F. et al. (2005) Neuropathological findings in cats with clinically suspect but histologically unconfirmed feline spongiform encephalopathy. Veterinary Record 156. 472-477.

http://archive.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/bse/oth

3 further cheetah cases have occured, plus 1 lion, plus all the primates, and 20 additional house cats. Nothing has been published on any of these UK cases either. One supposes the problem here with publishing is that many unpublished cases were _born_ long after the feed "ban". Caught between a rock and a hard place: leaky ban or horizontal transmission (or both).

http://www.mad-cow.org/may99_zoo_news.html

http://www.mad-cow.org/00/aug00_late_news.html#ggg

Evidence That Transmissible Mink Encephalopathy Results from Feeding Infected Cattle

Over the next 8-10 weeks, approximately 40% of all the adult mink on the farm died from TME.

snip...

The rancher was a "dead stock" feeder using mostly (>95%) downer or dead dairy cattle...

http://collections.europarchive.org/tna/20090505194948/http://bseinqu

In Confidence - Perceptions of unconventional slow virus diseases of animals in the USA - APRIL-MAY 1989 - G A H Wells

3. Prof. A. Robertson gave a brief account of BSE. The US approach was to accord it a very low profile indeed. Dr. A Thiermann showed the picture in the "Independent" with cattle being incinerated and thought this was a fanatical incident to be avoided in the US at all costs. ...

http://web.archive.org/web/20060307063531/http://www.bseinquiry.go

Wednesday, September 23, 2015

NIH Availability for Licensing AGENCY: [FR Doc. 2015–24117 Filed 9–22–15; 8:45 am] Detection and Discrimination of Classical and Atypical L-Type BSE Strains by RT-QuIC

http://bovineprp.blogspot.com/2015/09/nih-availability-for-licensing-agency.html

Thursday, October 1, 2015

H-type bovine spongiform encephalopathy associated with E211K prion protein polymorphism: clinical and pathologic features in wild-type and E211K cattle following intracranial inoculation

Master Obi-Wan Kenobi, Kemosabe...THIS IS NOT GOOD GOOSE!...grasshopper...tonto...tss

http://bovineprp.blogspot.com/2015/10/h-type-bovine-spongiform-encephalopathy.html

Wednesday, May 27, 2015

BSE Case Associated with Prion Protein Gene Mutation

http://bovineprp.blogspot.com/2015/05/bse-case-associated-with-prion-protein.html

spontaneous atypical BSE ???

don't let anyone fool you. spontaneous TSE prion disease is a hoax in natural cases, never proven.

all one has to do is look at France. France is having one hell of an epidemic of atypical BSE, probably why they stopped testing for BSE, problem solved $$$ same as the USA, that's why they stopped testing for BSE mad cow disease in numbers they could find any with, after those atypical BSE cases started showing up. shut down the testing to numbers set up by OIE that are so low, you could only by accident find a case of BSE aka mad cow disease. and this brilliant idea by the WHO et al, to change the name of mad cow disease, thinking that might change things is preposterous. it's all about money now folks, when the OIE, USDA and everyone else went along and made the TSE prion disease aka mad cow type disease a legal trading commodity by the BSE MRR policy, I would say everyone bit off more then they can chew, and they will just have to digest those TSE Prions coming from North America, and like it, and just prey you don't get a mad cow type disease i.e. Transmissible Spongiform Encephalopathy TSE prion disease in the decades to come, and or pass it to some other poor soul via the iatrogenic medical surgical tissue friendly fire mode of transmission i.e. second hand transmission. it's real folks, just not documented much, due to lack of trace back efforts. all iatrogenic cjd is, is sporadic cjd, until the iatrogenic event is tracked down and documented, and put into the academic and public domain, which very seldom happens. ...

As of December 2011, around 60 atypical BSE cases have currently been reported in 13 countries, *** with over one third in France.

http://www.biomedcentral.com/1746-6148/8/74

atypical spontaneous BSE in France LOL

FRANCE STOPS TESTING FOR MAD COW DISEASE BSE, and here's why, to many spontaneous events of mad cow disease $$$

***so 20 cases of atypical BSE in France, compared to the remaining 40 cases in the remaining 12 Countries, divided by the remaining 12 Countries, about 3+ cases per country, besides Frances 20 cases. you cannot explain this away with any spontaneous BSe. ...TSS

Sunday, October 5, 2014

France stops BSE testing for Mad Cow Disease

http://transmissiblespongiformencephalopathy.blogspot.com/2014/10/stops-bse-testing-for-mad-cow.html

Saturday, September 12, 2015

The Canadian Management of Bovine Spongiform Encephalopathy in Historical and Scientific Perspective, 1990-2014

>>>We propose that Canadian policies largely ignored the implicit medical nature of BSE, treating it as a purely agricultural and veterinary issue. In this way, policies to protect Canadians were often delayed and incomplete, in a manner disturbingly reminiscent of Britain's failed management of BSE. Despite assurances to the contrary, it is premature to conclude that BSE (and with it the risk of variant Creutzfeldt-Jakob disease) is a thing of Canada's past: BSE remains very

much an issue in Canada's present. <<<

http://bovineprp.blogspot.com/2015/09/the-canadian-management-of-bovine.html

Thursday, September 10, 2015

25th Meeting of the Transmissible Spongiform Encephalopathies Advisory Committee Food and Drug Administration Silver Spring, Maryland June 1, 2015

http://tseac.blogspot.com/2015/09/25th-meeting-of-transmissible.html

U.S.A. 50 STATE BSE MAD COW CONFERENCE CALL Jan. 9, 2001

http://tseac.blogspot.com/2011/02/usa-50-state-bse-mad-cow-conference.html

http://madcowusda.blogspot.com/2012/02/eight-former-secretaries-of-agriculture.html

Monday, October 26, 2015

FDA PART 589 -- SUBSTANCES PROHIBITED FROM USE IN ANIMAL FOOD OR FEED VIOLATIONS OFFICIAL ACTION INDICATED OIA UPDATE October 2015

http://madcowusda.blogspot.com/2015/10/fda-part-589-substances-prohibited-from.html

.150: Zoonotic potential of L-type BSE prions: A new prion disease in humans?

Emilie Jaumain,1 Stéphane Haïk,2 Isabelle Quadrio,3 Laetitia Herzog,1 Fabienne Reine,1 Armand Perret-Liaudet,3 Human Rezaei,1 Hubert Laude,1 Jean-Luc Vilotte,4 and Vincent Béringue1 1INR A (Institut National de la Recherche Agronomique); UR892; Virologie Immunologie Moléculaires; Jouy-en-Josas, France; 2IN SERM; Equipe maladie d'Alzheimer et maladies à Prions; CRicm; UMRS 1127; CNR S; UPMC. R.; ICM, Hôpital de la Salpêtrière; Paris, France; 3Neurobiologie, CMRR , Gériatrie, Hospices Civils de Lyon, Université Lyon 1-CNR S UMR5292-IN SERM U1028; Lyon, France; 3INR A; UMR1313; Génétique Animale et Biologie Intégrative; Jouy-en-Josas, France

Two novel prion strains, referred to as BSE-L and BSE-H, have been recognized in bovines through active prion surveillance programs, both being distinct from the epizootic, 'classical', BSE strain (C-BSE). Both H and L-types have been detected worldwide as rare cases occurring in aged animals. Like C-BSE prions, H- and L-types prions can propagate with relative ease in foreign species or in transgenic mouse lines expressing heterologous PrP sequences. A prion exhibiting biological properties similar to C-BSE agent sometimes emerged from these cross-species transmissions. Previously, L-type prions were shown to transmit to transgenic mice expressing human PrP with methionine at codon 129 with higher efficacy than C-BSE prions. Here, we examined whether L-type prions propagate without any apparent transmission barrier in these mice and whether such 'humanised' L-type prions share biological properties with CJD prions. L-type prions and a panel of human CJD cases with various genotypes at codon 129 and electrophoretic PrPres signatures were serially transmitted by intracerebral route to human PrP mice. The biological phenotypes induced by these agents were compared by all the standard methods currently used to distinguish between prion strains. At each passage, L-type prions were also transmitted back to bovine PrP mice to assess whether the agent has evolved upon passaging on the human PrP sequence. L-type prions transmitted to human PrP mice at 100% attack rate, without notable alteration in the mean incubation times over 5 passages. At each passage, 'humanized' L-type prions were able to transmit back to bovine PrP transgenic mice without apparent transmission barrier, as based on the survival time and the restoration of a L-type BSE phenotype. Comparison of mean incubation times on primary and subsequent passages in human PrP mice showed no overlap between L-type and sporadic CJD agents. While the electrophoretic signature and regional distribution of PrPres in L-type diseased mouse brains resembled that seen after transmission of MM2 CJD

strain type, both agents exhibited distinct resistance of the associated PrPres molecules to protease denaturation.

In summary, L-type prions can be passaged on the human PrP sequence without any obvious transmission barrier. The phenotype obtained differs from the classical CJD prion types known so far. Careful extrapolation would suggest that the zoonotic transmission of this agent could establish a new prion disease type in humans.

========Prion2013==========

2012 ATYPICAL L-TYPE BASE BSE TSE PRION CALIFORNIA 'confirmed' Saturday, August 4, 2012

*** Final Feed Investigation Summary - California BSE Case - July 2012

http://transmissiblespongiformencephalopathy.blogspot.com/2012/08/feed-investigation-summary.html

31 Jan 2015 at 20:14 GMT

*** Ruminant feed ban for cervids in the United States? ***

Singeltary et al

31 Jan 2015 at 20:14 GMT

http://www.plosone.org/annotation/listThread.action?root=85351

*** Singeltary reply ; Molecular, Biochemical and Genetic Characteristics of BSE in Canada Singeltary reply ;

http://www.plosone.org/annotation/listThread.action;jsessionid=635CE
root=7143

*** It also suggests a similar cause or source for atypical BSE in these countries. ***

Discussion: The C, L and H type BSE cases in Canada exhibit molecular characteristics similar to those described for classical and atypical BSE cases from Europe and Japan.

*** This supports the theory that the importation of BSE contaminated feedstuff is the source of C-type BSE in Canada.

*** It also suggests a similar cause or source for atypical BSE in these countries. ***

see page 176 of 201 pages...tss

http://www.neuroprion.org/resources/pdf_docs/conferences/prion2009

CONCLUSIONS:

Characterisation of the causal agents of disease resulting from exposure of cattle to naturally occurring scrapie agents sourced in Great Britain did not reveal evidence of classical or atypical BSE, but did identify two distinct previously recognised strains of scrapie. Although scrapie was still recognizable upon cattle passage there were irreconcilable discrepancies between the results of biological strain typing approaches and molecular profiling methods, suggesting that the latter may not be appropriate for the identification and differentiation of atypical, particularly L-type, BSE agents from cattle experimentally infected with a potential mixture of classical scrapie strains from sheep sources.

PMID: 26205536 [PubMed - in process]

http://www.ncbi.nlm.nih.gov/pubmed/26205536

Conclusions

Two different disease phenotypes were produced after intracerebral inoculation of cattle with scrapie brain pools sourced pre-1975 and post-1990 in GB, which were not readily explained by any differences in PrP genotype of the cattle. Based on pathological and molecular characteristics and biological characterisation in bank voles and transgenic mice there was no clear evidence of an agent derived from the

cattle resembling classical or atypical forms of BSE. Transmissions in bank voles identified previously isolated scrapie strains and some similarities to the experimental isolate CH1641. Contrary to the transmission results in rodents, the results for the molecular techniques, which have been adopted for the detection of atypical BSE cases, suggest that they may not be appropriate for differentiating WB profiles in cattle following infection from an ovine scrapie source.

http://download.springer.com/static/pdf/216/art%253A10.1186%252F
015-1260-3.pdf?
originUrl=http%3A%2F%2Flink.springer.com%2Farticle%2F10.1186%
015-1260-
3&token2=exp=1438200472~acl=%2Fstatic%2Fpdf%2F216%2Fart%
015-1260-
3.pdf%3ForiginUrl%3Dhttp%253A%252F%252Flink.springer.com%2
015-1260-
3*~hmac=694bb62bfe474dfd8aa4a22c016539137454993dfe25d67

Monday, July 20, 2015

Does the Presence of Scrapie Affect the Ability of Current Statutory Discriminatory Tests To Detect the Presence of Bovine Spongiform Encephalopathy?

http://transmissiblespongiformencephalopathy.blogspot.com/2015/07/
presence-of-scrapie-affect-ability.html

Wednesday, July 29, 2015

Further characterisation of transmissible spongiform encephalopathy phenotypes after inoculation of cattle with two temporally separated sources of sheep scrapie from Great Britain

http://transmissiblespongiformencephalopathy.blogspot.com/2015/07/
characterisation-of.html

IBNC Tauopathy or TSE Prion disease, it appears, no one is sure

Singeltary et al

Posted by flounder on 03 Jul 2015 at 16:53 GMT

http://www.plosone.org/annotation/listThread.action?
root=86610

Monday, October 26, 2015

FDA PART 589 -- SUBSTANCES PROHIBITED FROM USE IN ANIMAL FOOD OR FEED VIOLATIONS OFFICIAL ACTION INDICATED OIA UPDATE October 2015

http://madcowusda.blogspot.com/2015/10/fda-part-589-
substances-prohibited-from.html

Thursday, July 24, 2014

Protocol for further laboratory investigations into the distribution of infectivity of Atypical BSE SCIENTIFIC REPORT OF EFSA

http://www.efsa.europa.eu/sites/default/files/scientific_output/files/mair

http://bse-atypical.blogspot.com/2014/07/protocol-for-further-
laboratory.html

Wednesday, October 30, 2013

SPECIFIED RISK MATERIAL (SRM) CONTROL VERIFICATION TASK FSIS NOTICE 70-13 10/30/13

http://madcowusda.blogspot.com/2013/10/specified-risk-
material-srm-control.html

Review Methods for Differentiating Prion Types in Food-Producing Animals

Kevin C. Gough 1,*, Helen C. Rees 2, Sarah E. Ives 1 and Ben C. Maddison 2 1 School of Veterinary Medicine and Science, The University of Nottingham, Sutton Bonington Campus,

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Abstract: Prions are an enigma amongst infectious disease agents as they lack a genome yet confer specific pathologies thought to be dictated mainly, if not solely, by the conformation of the disease form of the prion protein (PrPSc). Prion diseases affect humans and animals, the latter including the food-producing ruminant species cattle, sheep, goats and deer. Importantly, it has been shown that the disease agent of bovine spongiform encephalopathy (BSE) is zoonotic, causing variant Creutzfeldt Jakob disease (vCJD) in humans. Current diagnostic tests can distinguish different prion types and in foodproducing animals these focus on the differentiation of BSE from the non-zoonotic agents. Whilst BSE cases are now rare, atypical forms of both scrapie and BSE have been reported, as well as two types of chronic wasting disease (CWD) in cervids. Typing of animal prion isolates remains an important aspect of prion diagnosis and is now becoming more focused on identifying the range of prion types that are present in food-producing animals and also developing tests that can screen for emerging, novel prion diseases. Here, we review prion typing methodologies in light of current and emerging prion types in food-producing animals.

snip...

8. Conclusions and Future Perspectives Whilst the cases of BSE in ruminants is now very low and the associated concern for the contamination of the human food chain with the zoonotic BSE agent has eased, there are still concerns surrounding the exposure of humans to prions from food-producing animals. The more recent description of atypical/Nor98 scrapie in goats/sheep and atypical bovine BSE as well as the discovery of two distinct types of CWD all raise the possibility that further types of prions are circulating in ruminants that are not detected and/or defined by current assay methods. An additional concern is that novel types may emerge in these animals. One diagnostic challenge in prion biology is to develop and apply prion typing tests to fully elucidate the range of existing prion types in ruminants and to monitor for the emergence of novel types. This is a significant challenge as it is unknown what molecular and pathological differences any novel type will have compared to those already described. Therefore, assays that have a wide range of distinct measurements that describe a PrPSc type or in vivo pathology will be best suited for diagnosing new prion types...

snip...see full text ;

http://www.mdpi.com/2079-7737/4/4/785/htm

CWD

Decision on listing (new CH)

TAHSC & SCAD & AHG

Pending AHG

http://www.oie.int/fileadmin/Home/eng/Internationa_Standard_Setting/

The Director General also expressed his concerns on the impact of atypical BSE in disease surveillance notification and status recognition and requested the Commission to continue its work, in coordination with the Terrestrial Animal Health Standard Commission (Code Commission), to address this issue.

http://www.oie.int/fileadmin/Home/eng/Internationa_Standard_Setting/

applications from Member Countries for the recognition of BSE risk status of Member Countries. The ad hoc Group also amended Chapter 11.4. on bovine spongiform

encephalopathy to consider the impact of atypical BSE on the countries' BSE risk status and to clarify that the requirements for risk classification only relates to classical BSE.

The Commission recommended that the Assembly recognise the following Member Countries as having a negligible BSE risk: Cyprus, Czech Republic, France, Ireland, Liechtenstein and Switzerland.

The Commission also agreed with the conclusion of the ad hoc Group regarding the non-compliance of the application of a Member Country.

In addition the Commission discussed in depth the application from one Member Country and concurred with the ad hoc Group that a mission to the country would be recommended to come to an informed decision.

The Commission agreed with the modifications proposed by the ad hoc Group on Chapter 11.4. on BSE to differentiate atypical from classical BSE and to consider the impact of atypical BSE on BSE risk status and on public health.

The Commission acknowledged with appreciation the work done by the ad hoc Group to adapt the surveillance system to the current BSE incidence considering the role of both atypical and classical BSE. However, although scientifically robust, this model gave more weight to the surveillance in older animals and allocated higher surveillance points to those countries that focus their surveillance to aged animals. It appeared not to be appropriate to some of OIE Member Countries already recognised as having a controlled or negligible BSE risk status. The Commission concluded that the proposed modification could not be considered at this stage for inclusion in the Terrestrial Code.

The Commission suggested that the Biological Standard Commission consider a revision of the BSE chapter of the Terrestrial Manual to include the description of the available tests able to discriminate atypical from classical BSE.

The Commission took note of the recommendation of the ad hoc Group regarding terminology and agreed that defining technical terms could be beneficial and that it could be done at the next ad hoc Group meeting when the revision of the questionnaire would be on the agenda. However, the Commission did not consider that the proposal to translate some technical terms into more languages other than the OIE official languages was a priority.

The amended chapter and the report of the ad hoc Group were provided to the Code Commission for further processing.

The endorsed report of the ad hoc Group is attached as Annex 12.

Scientific Commission/February 2015

snip...

Annex 12

Original: English

November 2014

REPORT OF THE MEETING OF THE OIE AD HOC GROUP

ON BOVINE SPONGIFORM ENCEPHALOPATHY RISK STATUS EVALUATION OF MEMBER COUNTRIES

Paris, 25-27 November 2014

_____

A meeting of the ad hoc Group on bovine spongiform encephalopathy (BSE) risk status evaluation of Member Countries (hereafter the Group) was held at the OIE Headquarters from 25 to 27 November 2014.

1. Opening

On behalf of Dr Bernard Vallat, Director General of the OIE, Dr Brian Evans, the OIE Deputy Director General and Head of

Scientific and Technical Department, welcomed and thanked the experts for their commitment towards the OIE and for personal and professional time invested to evaluate the dossiers.

Dr Evans ensured the Group that the challenges met to assess the applications were fully recognised and that, to increasingly take on board the difficulty of the assessment, the OIE Director General supported the Scientific Commission for Animal Diseases proposing that more in-country missions be conducted to verify the information provided in the written dossiers.

He mentioned that three missions to Member Countries would be planned before the upcoming General Session, reflecting the considerable involvement of the Scientific Commission at the national and regional level to assist and meet the expectations of Member Countries. He also emphasised the importance of accountability and that the procedures should be consistently applied in a transparent manner and well-grounded with the Resolutions adopted by the World Assembly of Delegates.

Dr Evans informed the Group that a series of workshops would be conducted in the next two years in each of the OIE regions in order to provide training for Member Countries on the key elements to consider when preparing a dossier for official recognition of disease or risk status. The Group was informed that the pilot workshop would be conducted in the Americas focussing on BSE and classical swine fever. Therefore, the support and advice of the Group in the identification of problematic areas in the dossiers was requested.

Dr Evans noted that the Group would have to consider the global decline of BSE, the relative higher importance of atypical BSE, the human health impact and the cost of surveillance when revising the current BSE chapter of the Terrestrial Animal Health Code (Terrestrial Code).

He finally introduced Dr Kazutoshi Matsuo, who recently joined the Scientific and Technical Department. He would be engaged in the activities related to official status recognition.

2. Adoption of the agenda and appointment of chairperson and rapporteur

Dr Dagmar Heim was appointed Chair and Dr Martial Plantady acted as rapporteur with the support of the OIE Secretariat. The Group endorsed the proposed agenda. The agenda and list of participants are provided as Appendices I and II, respectively.

3. Evaluation of requests from Member Countries for the evaluation of BSE risk status

Preliminary analyses were conducted by two members of the Group for each dossier (as allocated by the OIE Headquarters) prior to the meeting. The experts presented their key findings to the plenary, which proceeded with in-depth discussion, dossier by dossier, on the applicant Member Countries' compliance with the provisions on BSE risk status in the Terrestrial Code. Where necessary, messages were sent electronically to the applicants requesting additional information. All Member Countries contacted provided the requested information to the Group on time.

Dr John Kellar could not attend the meeting but provided his feed-back on the dossiers and on the other topics of the agenda, before and during the meeting, through electronic correspondence. Furthermore, he participated in parts of the discussion via teleconference on 26 November 2014.

3.1. Cyprus

The Group recalled that in July 2007 the OIE received a dossier from Cyprus to evaluate the BSE risk status of its cattle population in accordance with the Terrestrial Code. The recommendation of the Group at that time was that Cyprus should be regarded as having met the requirements for

recognition as complying with the BSE Chapter of the Terrestrial Code as ‘controlled BSE risk’.

In September 2014, Cyprus submitted a dossier seeking a negligible BSE risk status. The Group agreed that the submission conformed to the guidelines circulated for Member Countries wishing to make a formal evaluation of their BSE risk status according to the requirements of the Terrestrial Code.

Points specifically noted by the Group were summarised in the following discussion.

a) Section 1: Risk Assessment — Article 11.4.2. point 1

 Risk assessment for entry of the BSE agent

The Group considered that the conclusion of the entry assessment was that the risk that the BSE agent could have entered Cyprus during the interval covered by the assessment, although very low, was not negligible.

 Risk of recycling and amplification of the BSE agent

The Group considered that the conclusion of the exposure assessment was that there was a negligible risk of recycling and amplification of the BSE agent if it were present in Cyprus’s cattle population during the interval covered by the assessment.

b) Surveillance according to Articles 11.4.20.-11.4.22.

The Group noted that the surveillance undertaken exceeded the minimum requirements of type B surveillance according to Article 11.4.22. on surveillance for BSE in the Terrestrial Code. 8,715 surveillance points were collected, compared to a minimal requirement of 3,300 for an adult cattle population of 31,918 over two years of age.

c) Other requirements — Article 11.4.2. points 2–4

 Awareness programme

The Group noted that the awareness programme started in 1991 and met the requirements of the Terrestrial Code.

 Compulsory notification and investigation

The Group noted that BSE was declared a notifiable disease under relevant legislation since 1990 and determined that the system for compulsory notification and investigation met the requirements of the Terrestrial Code.

 Laboratory examination

The Group determined that the arrangements for laboratory examination met the requirements of the Manual of Diagnostic Tests and Vaccines for Terrestrial Animals (Terrestrial Manual).

 Appropriate level of control and audit of the feed ban

The Group noted that the appropriate legislation, control and audit of the proper implementation of the feed ban had been in force for at least eight years.

d) BSE history in the country

No BSE case had been recorded in Cyprus.

e) Compliance with conditions for ‘negligible BSE risk’ status - Article 11.4.3.

Based on the information provided, the Group recommended that Cyprus be regarded as having met the requirements for recognition as complying with the BSE Chapter of the Terrestrial Code as ‘negligible BSE risk’.

f) Conclusions

 Recommended status: ‘Negligible BSE risk’.

3.2. Czech Republic

The Group recalled that in July 2007 the OIE received a dossier from Czech Republic to evaluate the BSE risk status of its cattle population in accordance with the Terrestrial Code. The recommendation of the Group at that time was that the Czech Republic should be regarded as having met the requirements for recognition as complying with the BSE Chapter of the Terrestrial Code as ‘controlled BSE risk’.

In September 2014, the Czech Republic submitted a dossier seeking a negligible BSE risk status. The Group agreed that the submission conformed to the guidelines circulated for Member Countries wishing to make a formal evaluation of their BSE risk status according to the requirements of the Terrestrial Code.

The Group requested additional information and received clarification from the Czech Republic. Points specifically noted by the Group were summarised in the following discussion.

a) Section 1: Risk Assessment — Article 11.4.2. point 1

 Risk assessment for entry of the BSE agent

The Group considered that the conclusion of the entry assessment was that the risk that the BSE agent could have entered the Czech Republic during the interval covered by the assessment, although very low, was not negligible.

 Risk of recycling and amplification of the BSE agent

The Group considered that the conclusion of the exposure assessment was that there was a negligible risk of recycling and amplification of the BSE agent if it were present in Czech Republic’s cattle population during the interval covered by the assessment.

b) Surveillance according to Articles 11.4.20.-11.4.22.

The Group noted that the surveillance undertaken exceeded the minimum requirements of type B surveillance according to Article 11.4.22. on surveillance for BSE in the Terrestrial Code. 207,356 surveillance points were collected, compared to a minimal requirement of 71,500 for an adult cattle population of 663,423 over two years of age.

c) Other requirements — Article 11.4.2. points 2–4

 Awareness programme

The Group determined that the awareness programme began in 1991 and met the requirements of the Terrestrial Code.

 Compulsory notification and investigation

The Group noted that BSE was declared a notifiable disease under relevant legislation since 1999 and determined that the system for compulsory notification and investigation met the requirements of the Terrestrial Code.

 Laboratory examination

The Group determined that the arrangements for laboratory examination met the requirements of the Terrestrial Manual.

 Appropriate level of control and audit of the feed ban

The Group noted that the appropriate legislation, control and audit of the proper implementation of the feed ban had been in force for at least eight years.

d) BSE history in the country

The Group noted that the Czech Republic had reported 30 cases of BSE. The youngest BSE case was born on 8 May

2004, meaning that all indigenous cases would have been born more than 11 years preceding the World Assembly in May 2015. Therefore, the Czech Republic had met the provisions of Article 11.4.3. point 3 b). All cattle which were reared with the BSE cases during their first year of life, and which investigation showed consumed the same potentially contaminated feed during that period, if alive in the country, were completely destroyed.

e) Compliance with conditions for 'negligible BSE risk' status - Article 11.4.3.

Based on the information provided, the Group recommended that the Czech Republic be regarded as having met the requirements for recognition as complying with the BSE Chapter of the Terrestrial Code as 'negligible BSE risk'.

f) Conclusions

 Recommended status: 'Negligible BSE risk'.

3.3. France

In accordance with the established procedures, the participating expert from France withdrew from the discussions on France's dossier by the Group.

The Group recalled that in July 2007 the OIE received a dossier from France to evaluate the BSE risk status of its cattle population in accordance with the Terrestrial Code. The recommendation of the Group at that time was that France should be regarded as having met the requirements for recognition as complying with the BSE Chapter of the Terrestrial Code as 'controlled BSE risk'.

In September 2014, France submitted a dossier seeking a negligible BSE risk status. The Group agreed that the submission conformed to the guidelines circulated for Member Countries wishing to make a formal evaluation of their BSE risk status according to the requirements of the Terrestrial Code.

The Group requested additional information and received clarification from France. Points specifically noted by the Group were summarised in the following discussion.

a) Section 1: Risk Assessment — Article 11.4.2. point 1

 Risk assessment for entry of the BSE agent

The Group considered that the conclusion of the entry assessment was that the risk that the BSE agent could have entered France during the interval covered by the assessment, although very low, was not negligible.

 Risk of recycling and amplification of the BSE agent

The Group considered that the conclusion of the exposure assessment was that there was a negligible risk of recycling and amplification of the BSE agent if it were present in France's cattle population during the interval covered by the assessment.

b) Surveillance according to Articles 11.4.20.-11.4.22.

The Group noted that the surveillance undertaken exceeded the minimum requirements of type B surveillance according to Article 11.4.22. on surveillance for BSE in the Terrestrial Code. 2,236,881 surveillance points were collected, compared to a minimal requirement of 150,000 for an adult cattle population of 10,269,158 over two years of age.

c) Other requirements — Article 11.4.2. points 2–4

 Awareness programme

The Group determined that the awareness programme began in the early 1990's and met the requirements of the Terrestrial Code.

 Compulsory notification and investigation

The Group noted that BSE was declared a notifiable disease under relevant legislation since 1990 and determined that the system for compulsory notification and investigation met the requirements of the Terrestrial Code.

 Laboratory examination

The Group determined that the arrangements for laboratory examination met the requirements of the Terrestrial Manual.

 Appropriate level of control and audit of the feed ban

The Group noted that the appropriate legislation, control and audit of the proper implementation of the feed ban had been in force for at least eight years.

d) BSE history in the country

The Group noted that France had reported 985 cases of BSE. The youngest BSE case was born in April 2004, meaning that all indigenous cases would have been born more than 11 years preceding the World Assembly in May 2015. Therefore, France had met the provisions of Article 11.4.3. point 3 b). All cattle which were reared with the BSE cases during their first year of life, and which investigation showed consumed the same potentially contaminated feed during that period, if alive in the country, were completely destroyed.

e) Compliance with conditions for 'negligible BSE risk' status - Article 11.4.3.

Based on the information provided, the Group recommended that France be regarded as having met the requirements for recognition as complying with the BSE Chapter of the Terrestrial Code as 'negligible BSE risk'.

f) Conclusions

 Recommended status: 'Negligible BSE risk'.

3.4. Ireland

The Group recalled that in July 2007 the OIE received a dossier from Ireland to evaluate the BSE risk status of its cattle population in accordance with the Terrestrial Code. The recommendation of the Group at that time was that Ireland should be regarded as having met the requirements for recognition as complying with the BSE Chapter of the Terrestrial Code as 'controlled BSE risk'.

In September 2014, Ireland submitted a dossier seeking a negligible BSE risk status. The Group agreed that the submission conformed to the guidelines circulated for Member Countries wishing to make a formal evaluation of their BSE risk status according to the requirements of the Terrestrial Code.

The Group requested additional information and received clarification from Ireland. Points specifically noted by the Group were summarised in the following discussion.

a) Section 1: Risk Assessment — Article 11.4.2. point 1

 Risk assessment for entry of the BSE agent

The Group considered that the conclusion of the entry assessment was that the risk that the BSE agent could have entered Ireland during the interval covered by the assessment, although very low, was not negligible.

 Risk of recycling and amplification of the BSE agent

The Group considered that the conclusion of the exposure assessment was that there was a negligible risk of recycling and amplification of the BSE agent if it were present in Ireland's cattle population during the interval covered by the

assessment.

b) Surveillance according to Articles 11.4.20.-11.4.22.

The Group noted that the surveillance undertaken exceeded the minimum requirements of type B surveillance according to Article 11.4.22. on surveillance for BSE in the Terrestrial Code. 584,475 surveillance points were collected, compared to a minimal requirement of 150,000 for an adult cattle population of 3,123,200 over two years of age.

c) Other requirements — Article 11.4.2. points 2–4

 Awareness programme

The Group determined that the awareness programme began in 1996 and met the requirements of the Terrestrial Code.

 Compulsory notification and investigation

The Group noted that BSE was declared a notifiable disease under relevant legislation since 1989 and determined that the system for compulsory notification and investigation met the requirements of the Terrestrial Code.

 Laboratory examination

The Group determined that the arrangements for laboratory examination met the requirements of the Terrestrial Manual.

 Appropriate level of control and audit of the feed ban

The Group noted that the appropriate legislation, control and audit of the proper implementation of the feed ban had been in force for at least eight years.

d) BSE history in the country

The Group noted that Ireland had reported 1659 cases of BSE. The youngest BSE case was born in April 2004, meaning that all indigenous cases would have been born more than 11 years preceding the World Assembly in May 2015.

Therefore, Ireland had met the provisions of Article 11.4.3. point 3 b). All cattle which were reared with the BSE cases during their first year of life, and which investigation showed consumed the same potentially contaminated feed during that period, if alive in the country, were completely destroyed.

e) Compliance with conditions for ‘negligible BSE risk’ status - Article 11.4.3.

Based on the information provided, the Group recommended that Ireland be regarded as having met the requirements for recognition as complying with the BSE Chapter of the Terrestrial Code as ‘negligible BSE risk’.

f) Conclusions

 Recommended status: ‘Negligible BSE risk’.

3.5. Liechtenstein

In accordance with the established procedures, the participating expert from Switzerland, expressing a possible conflict of interest, withdrew from the discussions on Liechtenstein’s dossier by the Group.

The Group recalled that in January 2008 the OIE received a dossier from Liechtenstein to evaluate the BSE risk status of its cattle population in accordance with the Terrestrial Code. The recommendation of the Group at that time was that Liechtenstein should be regarded as having met the requirements for recognition as complying with the BSE Chapter of the Terrestrial Code as ‘controlled BSE risk’.

In September 2014, Liechtenstein submitted a dossier seeking a negligible BSE risk status. The Group agreed that

the submission conformed to the guidelines circulated for Member Countries wishing to make a formal evaluation of their BSE risk status according to the requirements of the Terrestrial Code.

Points specifically noted by the Group were summarised in the following discussion.

a) Section 1: Risk Assessment — Article 11.4.2. point 1

 Risk assessment for entry of the BSE agent

The Group considered that the conclusion of the entry assessment was that the risk that the BSE agent could have entered Liechtenstein during the interval covered by the assessment, although very low, was not negligible.

 Risk of recycling and amplification of the BSE agent

The Group considered that the conclusion of the exposure assessment was that there was a negligible risk of recycling and amplification of the BSE agent if it were present in Liechtenstein's cattle population during the interval covered by the assessment.

b) Surveillance according to Articles 11.4.20.-11.4.22.

The Group noted that the surveillance undertaken met the minimum requirements of type B surveillance according to Article 11.4.22. on surveillance for BSE in the Terrestrial Code. 399 surveillance points were collected, compared to a minimal requirement of 300 for an adult cattle population of 3.500 over two years of age. The Group also acknowledged the close interrelationship between Liechtenstein's and Switzerland's Veterinary Services.

c) Other requirements — Article 11.4.2. points 2–4

 Awareness programme

The Group determined that the awareness programme began in 1989 and met the requirements of the Terrestrial Code.

 Compulsory notification and investigation

The Group noted that BSE was declared a notifiable disease under relevant legislation since 1990 and determined that the system for compulsory notification and investigation met the requirements of the Terrestrial Code.

 Laboratory examination

The Group determined that the arrangements for laboratory examination met the requirements of the Terrestrial Manual.

 Appropriate level of control and audit of the feed ban

The Group noted that the appropriate legislation, control and audit of the proper implementation of the feed ban had been in force for at least eight years.

d) BSE history in the country

The Group noted that Liechtenstein had reported two cases of BSE. The youngest birth cohort reported as affected by BSE was born in 1993, meaning that all indigenous cases were born more than 11 years preceding the submission of the dossier. Therefore, Liechtenstein had met the provisions of Article 11.4.3. point 3 b). All cattle which were reared with the indigenous BSE cases during their first year of life, and which investigation showed consumed the same potentially contaminated feed during that period, if alive in the country, were completely destroyed.

e) Compliance with conditions for 'negligible BSE risk' status - Article 11.4.3.

Based on the information provided, the Group recommended that Liechtenstein be regarded as having met the

requirements for recognition as complying with the BSE Chapter of the Terrestrial Code as ‘negligible BSE risk’.

f) Conclusions

 Recommended status: ‘Negligible BSE risk’.

3.6. Switzerland

In accordance with the established procedures, the participating expert from Switzerland withdrew from the discussions on Switzerland’s dossier by the Group.

The Group recalled that in January 2007 the OIE received a dossier from Switzerland to evaluate the BSE risk status of its cattle population in accordance with the Terrestrial Code. The recommendation of the Group at that time was that Switzerland should be regarded as having met the requirements for recognition as complying with the BSE Chapter of the Terrestrial Code as ‘controlled BSE risk’.

In September 2014, Switzerland submitted a dossier seeking a negligible BSE risk status. The Group agreed that the submission conformed to the guidelines circulated for Member Countries wishing to make a formal evaluation of their BSE risk status according to the requirements of the Terrestrial Code.

Points specifically noted by the Group were summarised in the following discussion.

a) Section 1: Risk Assessment — Article 11.4.2. point 1

 Risk assessment for entry of the BSE agent

The Group considered that the conclusion of the entry assessment was that the risk that the BSE agent could have entered Switzerland during the interval covered by the assessment, although very low, was not negligible.

 Risk of recycling and amplification of the BSE agent

The Group considered that the conclusion of the exposure assessment was that there was a negligible risk of recycling and amplification of the BSE agent if it were present in Switzerland’s cattle population during the interval covered by the assessment.

b) Surveillance according to Articles 11.4.20.-11.4.22.

The Group noted that the surveillance undertaken exceeded the minimum requirements of type B surveillance according to Article 11.4.22. on surveillance for BSE in the Terrestrial Code. 104,961 surveillance points were collected, compared to a minimal requirement of 95,350 for an adult cattle population of 830,000 over two years of age.

c) Other requirements — Article 11.4.2. points 2–4

 Awareness programme The Group determined that the awareness programme began in 1989 and met the requirements of the Terrestrial Code.

 Compulsory notification and investigation

The Group noted that BSE was declared a notifiable disease under relevant legislation since 1990 and determined that the system for compulsory notification and investigation met the requirements of the Terrestrial Code.

 Laboratory examination

The Group determined that the arrangements for laboratory examination met the requirements of the Terrestrial Manual.

 Appropriate level of control and audit of the feed ban

The Group noted that the appropriate legislation, control and audit of the proper implementation of the feed ban had been in

force for at least eight years.

d) BSE history in the country

The Group noted that Switzerland had reported 467 cases of BSE. The youngest birth cohort reported as affected by BSE was born in 2003, meaning that all indigenous cases were born more than 11 years preceding the submission of the dossier. The Group acknowledged that the last BSE-case diagnosed as atypical BSE in Switzerland in 2012 and born in 2005, was imported from Germany at the age of 17 months. Therefore, Switzerland had met the provisions of Article 11.4.3. point 3 b). All cattle which were reared with the indigenous BSE cases during their first year of life, and which investigation showed consumed the same potentially contaminated feed during that period, if alive in the country, were completely destroyed.

e) Compliance with conditions for 'negligible BSE risk' status - Article 11.4.3.

Based on the information provided, the Group recommended that Switzerland be regarded as having met the requirements for recognition as complying with the BSE Chapter of the Terrestrial Code as 'negligible BSE risk'.

f) Conclusions

 Recommended status: 'Negligible BSE risk'.

3.7. Other Member Country requests

The Group assessed two additional requests from Member Countries for the recognition of their BSE risk status. One did not meet the requirements of the Terrestrial Code and the dossier was referred back to the corresponding Member Country. For the second, the Group recommended that a mission be conducted to the corresponding Member Country to verify compliance with Chapter 11.4. of the Terrestrial Code.

4. Revision of Chapter 11.4. of the Terrestrial Animal Health Code on BSE to consider atypical BSE

The Group summarised current scientific knowledge on several key questions to examine whether and how atypical BSE should be considered in Chapter 11.4. of the Terrestrial Code on BSE. The Group referred to its discussion of atypical BSE two years before, available in the report of its November 2012 meeting and invited the Scientific Commission and Member Countries to reflect on its content.

As the outcome of its discussion, the Group agreed that atypical BSE should be differentiated from classical BSE in Chapter 11.4. of the Terrestrial Code on BSE including its impact on BSE risk status recognition, maintenance and associated surveillance.

With regard to Article 11.4.25. the Group agreed that import of ruminants other than cattle is not considered to be a risk and therefore proposed to replace ruminant by cattle or bovine in the entire chapter, except in reference to the ruminant-to-ruminant feed ban. The suggested change is consistent with the chapter's evolution of focus from ruminants to cattle in preceding iterations.

The chapter was modified as follows:

Article 11.4.1.: General provisions and safe commodities

The Group clarified that the recommendations of the chapter cover both atypical and classical BSE.

Article 11.4.2.: The BSE risk status of the cattle population of a country, zone or compartment

The Group agreed that atypical BSE has to be considered to occur at the same rare background prevalence in any given cattle population. Therefore, the Group specified that the risk assessment should consider the potential factors for classical

BSE occurrence.

Entry assessment: the presence or absence of classical BSE agent should be carefully considered. The Group therefore proposed to slightly change Point 1 a i).

Exposure assessment: considering the probable rare background prevalence of atypical BSE in every indigenous bovine population, the Group emphasised that the exposure assessment be performed in every case, irrespective of the entry assessment.

Surveillance: in light of the implications for surveillance forthcoming from the declining tail of the classical BSE epidemic and the resulting, growing relative importance of atypical BSE, the Group acknowledged that the surveillance system should be revised in depth, including the potential reinstatement of a single surveillance goal per mature cattle population size.

The Group acknowledged that the current version of the Terrestrial Manual did not provide information on the suitable tests to be used to discriminate atypical from classical BSE. The Group recommended that Member Countries substantiate their findings with the support of the OIE Reference Laboratories for BSE.

Therefore the Group suggested the Scientific Commission to discuss with the Biological Standard Commission whether a revision of the BSE chapter of the Terrestrial Manual would be needed to consider tests able to discriminate atypical from classical BSE.

Article 11.4.3.: Negligible BSE risk

The Group agreed that the occurrence of atypical BSE cases (irrespective of age or birth year) should not influence official risk status, as long as the criteria of Article 11.4.2. have been complied with and an appropriate level of control gives evidence that the ruminant-to-ruminant feed ban has been efficient for the last eight years. The Group clarified that the follow-up of the cohorts of BSE cases was not applicable to atypical BSE cases since atypical BSE is not linked to feed practices.

Article 11.4.4.: Controlled BSE risk

The Group proposed changes similar to those proposed in Article 11.4.3.

Article 11.4.7.: Recommendations for the importation of cattle from a country, zone or compartment posing a negligible BSE risk but where there has been an indigenous case In accordance with the above changes, the Group clarified that these recommendations would be valid for a country, zone or compartment having a negligible BSE risk but only where there has been an indigenous case of classical BSE.

Article 11.4.9.: Recommendations for the importation of cattle from a country, zone or compartment posing an undetermined BSE risk

The Group proposed changes in line with those proposed in Article 11.4.3.

Article 11.4.10.: Recommendations for the importation of meat and meat products from a country, zone or compartment posing a negligible BSE risk

The Group clarified that the requirement in point 3 applies to countries with negligible BSE risk and one or more indigenous cases of classical BSE.

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The Group also considered the risk posed by atypical BSE and proposed a recommendation ensuring that the products were not contaminated with tissues listed in the newly proposed point 4 of Article 11.4.14. (brain, eye, spinal cord and skull from cattle aged more than 96 months).

Article 11.4.13.: Recommendations on ruminant-derived meat-and-bone meal or greaves

The Group discussed the risk of trading of ruminant MBM, acknowledging that atypical BSE was likely to exist in every domestic cattle population. Considering that the feed ban is the most important mitigating measure to avoid recycling, the Group proposed for countries with recognised negligible risk status for BSE, with or without reported cases, consideration that trade of ruminant MBM be restricted to cattle born after the effective enforcement of the ruminant-to-ruminant feed ban.

Article 11.4.14.: Recommendations on commodities that should not be traded

Considering that atypical BSE was likely to exist in any bovine population and the age distribution of atypical BSE cases, the Group recommended that brain, eye, spinal cord and skull not be traded if originated from cattle over 96 months (eight years) from negligible BSE risk countries and added a paragraph to this article.

Article 11.4.16.: Recommendations for the importation of tallow (other than as defined in Article 11.4.1.) intended for food, feed, fertilisers, cosmetics, pharmaceuticals including biologicals, or medical devices and Article 11.4.18.:

Recommendations for the importation of tallow derivatives (other than those made from tallow as defined in Article 11.4.1.) intended for food, feed, fertilisers, cosmetics, pharmaceuticals including biologicals, or medical devices

The changes in these two articles were proposed to reflect the proposal of Article 11.4.14.

Article 11.4.20.: Surveillance: introduction

The Group considered that the article was valid for both classical and atypical BSE.

Article 11.4.21.: Surveillance: description of cattle subpopulations

The Group estimated that the differentiation of classical and atypical BSE should be mentioned in the definition of the cattle sub-population.

Point 1: cattle over 30 months of age displaying behavioural or clinical signs consistent with BSE (clinical suspects).

The Group agreed that this should be limited to classical BSE as atypical cases do not show classical signs of BSE.

Article 11.4.22.: Surveillance activities

The Group agreed that the statistical model should be revised in depth to consider the evolution of classical BSE epidemiology, as well as the specificities of atypical surveillance.

The Group highlighted the following points to be taken into account in the potential revision of the statistical BSE model:

- Recalculation of the relative risk of positive BSE test by age and population stream

- Retention of current target populations

- Appropriateness of current target prevalence and merits of one type of surveillance ;

- Confidence level retention at 95%;

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- Design prevalence of at least one case per 100,000 in the adult population (current type A surveillance);

- Re-weighting of focus in favour of older animals

- Retention of sampling in younger animals;

- European Commission and OIE databases of all tested animals, including positives and their ages.

The Group agreed to revise the model for BSE surveillance in the weeks following the meeting and proposed a new model based on an estimation of the BSE incidence. Unfortunately the BSE surveillance point values of the proposed model were not applicable to all OIE Member Countries already having a BSE risk status.

The Group acknowledged that the seven years of surveillance was referring to the 95th percentile of the incubation period of classical BSE. However, considering the need of continuous surveillance, the Group agreed to maintain the possibility to accumulate points over the years. The proposal of seven years was kept with relation to classical BSE. However, the Group agreed that this may need to be revised once the incubation period of atypical BSE is known.

Article 11.4.23: BSE risk assessment: introduction

Changes were proposed to reflect the proposal of Article 11.4.2. and to clarify the wording.

Article 11.4.24.: The potential for the entry of the BSE agent through the importation of meat-and-bone meal or greaves
The Group adjusted the text in line with the proposed changes of previous articles.

Article 11.4.25.: The potential for the entry of the BSE agent through the importation of live animals potentially infected with BSE

The Group updated the article by clarifying that import of ruminants other than cattle is not considered to be a risk, and that import of cattle could present a risk of entry of the classical BSE agent when coming from countries with classical BSE.

The Group deleted the reference to hypothetical maternal transmission as its epidemiological significance has been downplayed.

Article 11.4.26.: The potential for the entry of the BSE agent through the importation of products of animal origin potentially infected with BSE

In addition to the changes already proposed in previous article, the Group proposed to delete the reference to feeding practices in dairy cows due to the imposed feed ban.

The Group also removed the reference to the length of time the animals lived in a country as this article is related to import of products and not of live animals. However, the Group clarified that these products should not encompass tissues known to contain BSE infectivity.

Article 11.4.27.: The potential for the exposure of cattle to the BSE agent through consumption of meat-and-bone meal or greaves of ruminant origin

Safe commodities being specifically listed in Article 11.4.1., the Group considered it inadequate to keep reference only to milk and blood in this article.

Article 11.4.28.: The origin of animal waste, the parameters of the rendering processes and the methods of animal feed production

The Group agreed to delete the first four bullet points that were not relevant for the purpose of this article.

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The Group considered that reticulo-endothelial tissues could not be included in the list of tissues where BSE agent is present at much higher titres and removed it to consider as specified risk material (SRM) only the central nervous system.

Article 11.4.29.: Conclusions of the risk assessment

The Group clarified the risk linked to classical BSE and to atypical BSE.

5. Consideration of the information provided by authors of the BSurvE model with regards to its update

The Group acknowledged that the authors of the BSurvE model could not update the model despite the need to consider the global evolution of BSE.

6. General considerations and advice to be provided to future applicant Member Countries

On a structural aspect, the Group strongly recommended that applicant Member Countries adhere to the questionnaire of Article 1.6.5. of the Terrestrial Code, answering all questions clearly and concisely. While acknowledging the importance of some appendices, the Group recalled that the central points should be covered in the core document. Appendices should be clearly cross-referenced in the dossier and their titles should provide the key words of their contents.

The Group would appreciate the presentation of a short summary/conclusion at the end of each section.

The Group clarified that full regulatory texts are not needed in the dossier but that a summary of the important regulatory texts should be provided to help the experts to understand the national situation.

The Group would expect that Member Countries, conducting visual inspection in feed mills and renderers, would identify apparent infractions, and would include explanations on the follow-up procedure applied to rule cross-contamination out.

The Group noted that the measures to mitigate the exposure assessment (feed ban, SRM removal, cross-contamination controls) were often not sufficiently detailed in the dossiers.

The Group was requested to provide advice on relevant points that should be covered in the workshop on the OIE procedure for the official recognition of country status, with reference to BSE. The Group identified the following as points of the questionnaire that would need explanations from the trainers:

- The definition and difference between feed mills and rendering plants

- The tables of the questionnaire related to the feed ban

- The surveillance point system

- The collection of the data and diagnostic protocols

The Group suggested that the trainers put the participants in the role of reviewers. A possibility could be to present blind extracts from several dossiers and request their opinions.

The Group also mentioned the potential merit in revisiting the entire cycle of BSE infectivity concurrently identifying where the risks are and where associated inspections are needed.

7. Other matters

The Group recommended that the Scientific Commission schedules the revision of the questionnaire (Article 1.6.5. of the Terrestrial Code). The Group suggested that such revision considers:

- Weighting of the exposure assessment with relation to the global evolution of BSE epidemiology;

- Definition of technical words such as rendering plants, feed mills, cohort;

- Inclusion of questions related to the capability of the Veterinary Services and disease notification.

Considering the difficulties faced in the evaluation of some

dossiers as a result of translation issues, the Group suggested that some technical terms (such as those of the Glossary) be translated into more languages than the OIE official languages such as Russian, Arabic, Chinese, Portuguese and Japanese.

8. Finalisation and adoption of the draft report

The Group reviewed and amended the draft report provided by the rapporteur. The Group agreed that the report reflected the discussions. _____

…/Appendices

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Appendix I

MEETING OF THE OIE AD HOC GROUP ON BOVINE SPONGIFORM ENCEPHALOPATHY (BSE) RISK STATUS EVALUATION OF MEMBER COUNTRIES

Paris, 25-27 November 2014

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Agenda

1. Opening

2. Adoption of the agenda and appointment of chairperson and rapporteur

3. Evaluation of applications from Member Countries for official recognition of BSE risk status

- Cyprus

- Ireland

- Czech Republic

- Liechtenstein

- France

- Switzerland

4. Revision of Chapter 11.4. of the Terrestrial Animal Health Code on BSE to consider atypical BSE

5. Consideration of the information provided by authors of the BSurvE model with regards to its update

6. General considerations and advices to be provided to future applicant Member Countries

7. Other matters

8. Finalisation and adoption of the draft report

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Appendix II

MEETING OF THE OIE AD HOC GROUP ON BOVINE SPONGIFORM ENCEPHALOPATHY (BSE) RISK STATUS EVALUATION OF MEMBER COUNTRIES

Paris, 25-27 November 2014

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http://www.oie.int/fileadmin/Home/eng/Internationa_Standard_Setting/

Sunday, October 18, 2015

*** World Organisation for Animal Health (OIE) and the Institut Pasteur Cooperating on animal disease and zoonosis research ***

http://bovineprp.blogspot.com/2015/10/world-organisation-for-animal-health.html

Saturday, September 19, 2015

Wednesday, September 23, 2015

NIH Availability for Licensing AGENCY: [FR Doc. 2015–24117 Filed 9–22–15; 8:45 am] Detection and Discrimination of Classical and Atypical L-Type BSE Strains by RT-QuIC

http://bovineprp.blogspot.com/2015/09/nih-availability-for-licensing-agency.html

Friday, December 5, 2014

*** SPECIAL ALERT The OIE recommends strengthening animal disease surveillance worldwide OIE

BSE TSE PRION AKA MAD COW DISEASE ? ''the silence was deafening'' ...tss

http://transmissiblespongiformencephalopathy.blogspot.com/2014/12/alert-oie-recommends.html Monday, May 05, 2014

Member Country details for listing OIE CWD 2013 against the criteria of Article 1.2.2., the Code Commission recommends consideration for listing

http://chronic-wasting-disease.blogspot.com/2014/05/member-country-details-for-listing-oie.html

Thursday, December 20, 2012

OIE GROUP RECOMMENDS THAT SCRAPE PRION DISEASE BE DELISTED AND SAME OLD BSe WITH BOVINE MAD COW DISEASE

http://transmissiblespongiformencephalopathy.blogspot.com/2012/12/group-recommends-that-scrape-prion.html

Wednesday, February 16, 2011

IN CONFIDENCE

SCRAPIE TRANSMISSION TO CHIMPANZEES

IN CONFIDENCE

http://scrapie-usa.blogspot.com/2011/02/in-confidence-scrapie-transmission-to.html

Tuesday, July 14, 2009 U.S.

*** Emergency Bovine Spongiform Encephalopathy Response Plan Summary and BSE Red Book

Date: February 14, 2000 at 8:56 am PST

WHERE did we go wrong $$$

http://madcowtesting.blogspot.com/2009/07/us-emergency-bovine-spongiform.html Monday, November 30, 2009

*** USDA AND OIE COLLABORATE TO EXCLUDE ATYPICAL SCRAPIE NOR-98 ANIMAL HEALTH CODE, DOES NOT SURPRISE ME $

http://nor-98.blogspot.com/2009/11/usda-and-oie-collaborate-to-exclude.html

Tuesday, January 1, 2008

BSE OIE USDA

Subject: OIE BSE RECOMMENDATION FOR USA, bought and paid for by your local cattle dealers i.e. USDA

Date: May 14, 2007 at 9:00 am PST

OIE BSE RECOMMENDATION FOR USA, bought and paid for by your local cattle dealers i.e. USDA

STATEMENT BY DR. RON DEHAVEN REGARDING OIE RISK RECOMMENDATION

March 9, 2007

http://madcowtesting.blogspot.com/2008/01/bse-oie-usda.html

Thursday, October 22, 2015

Former Ag Secretary Ann Veneman talks women in agriculture and we talk mad cow disease USDA and what really happened

http://madcowusda.blogspot.com/2015/10/former-ag-

secretary-ann-veneman-talks.html

Tuesday, July 14, 2009 U.S.

*** Emergency Bovine Spongiform Encephalopathy Response Plan Summary and BSE Red Book

Date: February 14, 2000 at 8:56 am PST

WHERE did we go wrong $$$

http://madcowtesting.blogspot.com/2009/07/us-emergency-bovine-spongiform.html

*** Qualitative Analysis of BSE Risk Factors in the United States

February 13, 2000 at 3:37 pm PST (BSE red book)

http://bseusa.blogspot.com/2008/08/qualitative-analysis-of-bse-risk.html

Comments on technical aspects of the risk assessment were then submitted to FSIS.

Comments were received from Food and Water Watch, Food Animal Concerns Trust (FACT), Farm Sanctuary, R-CALF USA, Linda A Detwiler, and Terry S. Singeltary.

This document provides itemized replies to the public comments received on the 2005 updated Harvard BSE risk assessment. Please bear the following points in mind:

http://www.fsis.usda.gov/PDF/BSE_Risk_Assess_Response_Public_

Owens, Julie

From: Terry S. Singeltary Sr. [flounder9@verizon.net]

Sent: Monday, July 24, 2006 1:09 PM

To: FSIS RegulationsComments

Subject: [Docket No. FSIS-2006-0011] FSIS Harvard Risk Assessment of Bovine Spongiform Encephalopathy (BSE)

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(Adopted by the International Committee of the OIE on 23 May 2006)

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